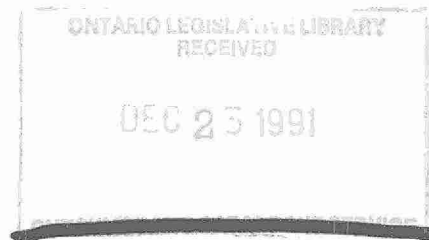


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# ENVIRONMENTAL RESEARCH

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DOSE RESPONSE FOR SELECTED ENVIRONMENTAL  
AIR POLLUTANTS:

A STUDY ON RUNNERS

RAC Report # 219G

Report prepared by:

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Report prepared for:

Research and Technology Branch  
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Dose Response for Selected Environmental  
Air Pollutants: A Study on Runners

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Final Report

ABSTRACT

Exercise increases respiratory minute ventilation, while altering mode of breathing (mouth vs. nose). It therefore can increase the dose of inhaled pollutants. Thus the risk associated with air pollution may be increased in people who exercise outdoors, particularly in urban environments. This study examined the effects of air pollution on runners during outdoor training runs in downtown Toronto from 1986-1988.

Subjects were selected from the Longboat Roadrunners Club, a local competitive group, which carries out weekly training runs of 6-10 miles (10-16 km) in downtown Toronto and along the Lakeshore transportation corridor during rush hour. Pulmonary function and an oxygen rebreathing estimate of blood carboxyhaemoglobin level (COHb%) were obtained before and after 75 training runs involving 70 athletes. Subjective reports of symptoms and an estimate of personal exposure to air pollution over the run were assessed. Local pollutant measurements including carbon monoxide (CO), nitrogen dioxide (NO<sub>2</sub>), sulphur dioxide and respirable suspended particulate matter, along with dry bulb temperature and relative humidity were made using portable multipollutant samplers carried on bicycles that accompanied the runners, thus giving an estimate of personal exposure. Exposure data were supplemented by data collected by the Ontario Ministry of the Environment (MOE) at two of their nearby fixed-site monitoring stations.

Symptoms were related to temperature, relative humidity and local concentrations of several of the pollutants. The runner's subjective assessment of exposure was found to be related jointly to COHb%, NO<sub>2</sub> and temperature. In turn, COHb% was found to be related to both CO and NO<sub>2</sub>, thereby establishing a reasonable causal link between exposure to an exhaust-related pollutant (indicated by CO), biological dose (COHb%) and the effect upon air pollution ratings. Relationships of air pollutant concentrations to pulmonary function were not as strong as to subjective estimates of pollution and to symptom ratings. The relationship of the subjective assessment of pollution exposure to CO and NO<sub>2</sub> concentrations was stronger when the concentrations were measured with the portable multipollutant samplers, than when measured at a nearby MOE fixed-site monitoring station, emphasizing the importance of assessing local pollutant exposure.



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## INTRODUCTION

The cardio-respiratory benefits of exercise have been well documented. However, there has been less attention directed towards the risk potential for adverse health effects of inhaled pollutants while exercising outdoors, particularly in urban environments.

Major pollutant sources in urban areas include both industry and vehicular exhaust, producing primary pollutants (e.g. sulphur dioxide, carbon monoxide, nitric oxide and particulates) and secondary pollutants resulting from reactions of primary pollutants, ultraviolet light and other ambient chemical compounds (e.g. ozone, nitrogen dioxide, peroxyacetyl nitrate and aldehydes).

Although pollutant concentration is of primary concern when examining the health effects of exercising outdoors, other factors may exacerbate the response. The respiratory minute ventilation is proportional to the intensity of exercise or work rate (below ventilatory threshold), thus at higher work rates the pollutant dose (pollutant concentration x respiratory minute ventilation) is increased over the resting condition. When estimating pollutant dose, a third factor must also be considered, that is, exercise duration. Furthermore, at respiratory ventilations above 35 L/min, people tend to breathe oronasally, thus partially bypassing the normal filtration mechanism of the nose. Other factors

which can influence the response include environmental factors such as temperature and humidity, and physiological factors such as age (children, adults, elderly), fitness level (social exerciser vs competitive athlete), and the presence of underlying disease, particularly respiratory disease, as well as tobacco smoking.

Although many of the health effects produced by ambient levels of pollutants are relatively small, they may be of serious consequence to the performance of the elite competitive athlete, whose performance is timed to the millisecond. However, one must not overlook the recreational exercisers, in whom potential high risk groups include the elderly, children and those with underlying cardio-respiratory disease. Therefore, in assessing the effects of air pollutants on exercising individuals, consideration must be given to 1) the ambient air conditions (type and concentration of pollutants), 2) the intensity and duration of exercise being undertaken, 3) environmental factors, and 4) the age, level of fitness and presence of any underlying disease of the person exercising. However, air pollution mixtures are very complex, varying in both constituents and levels at any given time. Many have not to date been demonstrated to have significant effects on the exercising individual, or have not been systematically examined with respect to their impact.

The impact of vehicle-related pollutants upon human

health and performance has been the subject of several major reviews (1-4). The problem is particularly acute for the city runner, because a high ventilation rate is often combined with close exposure to traffic; one recent U.S. study showed, for example, a threefold rise of blood carboxyhaemoglobin levels (from about 1.5% to 4.5%) as a consequence of running for only half an hour in rush-hour traffic (5). While such high levels of carboxyhaemoglobin are tolerated by heavy smokers, they probably play a major role in the burden of increased cardiovascular disease experienced by the smoker. Myocardial damage from acute and chronic exposure to moderate levels of carbon monoxide has been demonstrated in rabbits (6,7), while firefighters, automobile tunnel workers and vehicle inspectors all have an increased cardiovascular mortality and morbidity relative to controls (8-10). Furthermore, the duration of exercise needed to precipitate angina is shortened by carbon monoxide exposure (11).

Studies by the U.S. Environmental Protection Agency (12) and locally in Toronto (13) have further shown that ambient CO levels in cities are highest on freeways surrounded by tall buildings where the traffic density is high and speeds are low.

There has been an increase in the number of joggers and people competing in races and "fun runs" in urban areas, specifically Toronto. This may pose a health risk to the

joggers, due to pollutant exposure. Although the pollutant levels in Toronto are relatively low, the "jogger" or "runner" is breathing at a high ventilation and thus is exposed to a much greater dose of pollutant than when at rest (14, 15, 16). Also, the jogger may experience "pockets" or high areas of pollution along his/her jogging route. The consequences of this pollutant exposure may result in an impairment of cardiorespiratory function (17,18) and a decrease in performance. However, problems exist in the measurement of personal pollutant exposure, specifically in joggers, who may traverse a large area. Any uni-site and/or remote (e.g. central station) method of measuring the pollutant exposure may not be representative of the personal exposure level (19-21). A multi-site method of measuring pollutant levels would better approximate the personal exposure level. The best method would be a personal monitor, carried by the subject, or in close proximity to the subject, although size constraints limit the number of different pollutants that can be measured by one monitor while still remaining portable.

While much can be learnt about the toxicity of environmental contaminants from animal experimentation, there are substantial problems in transferring such data directly to human populations. Logic might thus suggest the experimental exposure of human volunteers, but there are obvious ethical problems in extending the range of exposure

to pollutant concentrations that are likely to cause a deterioration of health. Much of the information needed for the difficult decisions of air management must thus be collected by epidemiological analysis of "natural experiments."

Runners provide ideal subjects for such experiments, from several points of view. Firstly, their habit of sustained and vigorous exercise increases the dose of most pollutants by a factor of at least ten, increasing exposure (22,23). Secondly, they often exercise along busy auto-routes where the concentration of car exhaust pollutants is particularly high. Thirdly, exposure to most compounds is exacerbated by the fact that a high demand for oxygen forces them to breathe through the mouth, by-passing the normal filtration mechanism of the nose. Rapid ventilation carries both particles and vapours deep into the lungs. Finally, their performance is timed to a small fraction of a second, so that the functional impact of pollution upon oxygen transport can be determined very precisely. Not only are such runners interesting from an epidemiological point of view, but given the mass interest in "fun-runs", any adverse consequences of such increased exposure to pollutants must be a matter of public concern.

An important feature of this study was the measure of personal exposure using multi-site mobile monitoring along the running route, to follow pollution levels over the course

of training runs. Other confounding factors including relative humidity and temperature have been introduced as co-variates in the analyses, thus controlling for such influences.

## METHODS

### 1) Subjects.

The difficulty in using many of the "fun-runs" in Toronto and elsewhere is that they attract a large population of runners with varied levels of ability, and that the group is not consistent in its composition from one meet to the next. We have thus tested members of the Longboat Roadrunners Club, to follow their performance on a weekly basis. This club, named after the internationally known Indian competitor from the Six Nations Reserve is a group of some 160 runners at different levels of fitness, who cover distances of up to 65 miles (110 km) per week. Some of the participants have achieved race times of 53 minutes for 10 miles (16 km); a ventilation of at least 80 L/min is to be anticipated (more than twice the threshold for oral breathing (24). On Wednesday evening (5:30 PM), their training and running route (6-10 miles) follows the downtown core and the crowded Lakeshore transportation corridor, where there is a potential for heavy exposure to vehicle exhaust.

A total of 81 subjects were entered into the study.

However, 10 subjects who only completed 1 test and one who had incomplete health information were not included in the analyses, thus leaving 70 subjects with 2 or more tests. Runners were tested a total of 2 to 20 times during Wednesday training runs (Table 1A). The runners fell largely into three groups, those that ran 6 miles (10 km), 8 miles (13 km) or 10 miles (16 km) during Wednesday training runs, although subjects may have run from 2 miles (3 km) to 14 miles (22.5 km) (Table 1C). Also, the subjects did not necessarily run the same distance each week.

To obtain a better assessment of air pollution exposure, a measure of dose was calculated by multiplying the integral of local pollutant concentration by running time (minutes) by ventilation rate (L/min). The runners recorded their heart rates by palpation at the mid-point of the run; this in turn was used to estimate their ventilation rates during the run. Runners were taken to the exercise lab at Toronto General Hospital, on a separate day from the run day, where they ran on a treadmill at a series of speeds covering their range of training and racing paces. Ventilation, heart rate and other variables were measured during the treadmill test. For each runner, a regression was obtained for heart rate vs. respiratory minute ventilation. In order to linearize the regression, the log ventilation was used. Thirty-nine runners had from 1 to 3 treadmill tests. Heart rates measured by the runners during training runs were used to



predict their ventilation, using the regression equation developed for each runner. Predicted ventilations obtained using the reported heart rates with the individual regression equations showed a mean  $\pm$  SD of  $68 \pm 27$  L/min. The estimated dose based on these predicted ventilations may be a better estimate of risk than the pollutant concentration taken alone, and may be used to account for any possible differences between the 6, 8 and 10 mile groups in pulmonary function and COHb% changes, or symptom ratings during training runs.

## 2. Air pollution exposure.

Field samplers (GAGE MULTI-POLLUTANT SAMPLERS) - SO<sub>2</sub>, NO<sub>2</sub> and respirable particulate matter were measured using sampling systems designed and constructed at The Gage Research Institute (25). We have used these samplers with success for the past several years (19, 25-28). They are small, portable, multi-pollutant samplers driven by a battery-operated pump system. Concentrations are reported as a time-weighted average over the sampling period. The particulate sampler consists of a cyclone and filter cassette assembly, operated at a flow rate of 1.7 L/min, yielding a 50% particle cut of 5 microns (29). Particulate filters are weighed to an accuracy of 0.001 mg and are desiccated (before weighing) in the presence of anhydrous calcium sulphate for 24 hrs, both before and after use. The filters

have been stored in closed petri dishes and could be analyzed at some future date for sulphates, nitrates, chlorides and trace metals at a later date. The gas systems for  $\text{NO}_2$  and  $\text{SO}_2$  each use 2 impingers in parallel (with one teflon inlet) containing appropriate absorbing solutions, operated at 1.0 L/min (500 ml/impinger).  $\text{SO}_2$  concentration is assessed by the West and Gaeke method (30), and the U.S. EPA NAAQS (31). The  $\text{NO}_2$  concentration is assessed by the TGS-ANSA method (32).

Samplers were carried on 2 bicycles (mobile samplers) to assess "personal exposure"; one bicycle followed alongside the 6 mile (10 km) runners and the other bicycle followed alongside the 10 mile (16 km) runners. Each bicycle carried 2 particulate samplers, 4  $\text{NO}_2$  impingers and 4  $\text{SO}_2$  impingers, as a check for intersampler accuracy. Gage stationary samplers were also located at a central MOE monitoring network site (Breadalbane) and included 2 particulate samplers, 2  $\text{NO}_2$  impingers and 2  $\text{SO}_2$  impingers.

The samplers were calibrated as in previous studies, with monthly calibration checks (26, 28).

CO Analyzer - CO concentrations were assessed using a Thermo Electron CO analyzer (Thermo Electron Inst., model 48). Carbon monoxide is detected by infra-red absorption. This is a highly selective method providing more stability and accuracy than electrochemical methods. The CO analyzer

was positioned at the start of the running route, where the runners gathered before the beginning of the run. It was used to measure CO for the carboxyhaemoglobin (COHb%) rebreathe test.

In order to assess "personal exposure", 2 portable CO monitors (Industrial Scientific, model CO-260) were used. These monitors are continuously reading, and use an electrochemical cell to detect CO. One monitor was carried on each bicycle. Recordings were taken at various sites along the route (Tables 3D and 3E).

The CO analyzers were zeroed and calibrated before each sampling period. The CO analyzers were calibrated using a primary standard CO cylinder gas. Calibration was also checked at the end of the sampling/testing period. Analyzers were checked for linearity on a monthly basis, using 3 primary standard CO cylinder gases with concentrations covering the range of 20, 40 and 60 ppm CO.

Collection of Other Environmental Data - Data were also obtained from the air pollution monitoring network of the Ontario Ministry of the Environment (temperature, O<sub>3</sub>, SO<sub>2</sub>, CO, total hydrocarbons, oxides of nitrogen, suspended particulate matter, lead, sulphates, nitrates, coefficient of haze and the Air Pollution Index), at their Breadalbane and Evans/Arnold sites. Temperature and relative humidity were obtained from Atmospheric Environment Services (AES), at their Trinity College site. The two MOE sites and the AES

site are shown on a map of the running route in Appendix I. The pollutant and environmental data were used along with the weekly Gage samplers' data to plot the weekly changes of the pollution along the running route taken by the Longboat Roadrunners Club every Wednesday evening. A temperature/humidity monitor (Jenway, model 5500) using a wet bulb/dry bulb system with air pump was carried on the Gage 10 mile bicycle and readings taken at 4 points along the route.

### 3) Health Effects.

Carboxyhaemoglobin Determinations (COHb%) - The COHb% was determined, pre and post run, by the rebreathing technique of Henderson and Apthorp (33). Subjects rebreathed from a 5 litre bag of 100% O<sub>2</sub> for 2 minutes, after which the bag was sampled for CO concentration. We have found this method to be reliable in previous studies (34, 35).

Pulmonary Function Measurements - Spirometric tests of lung function were made pre and post run using two Med-Science Wedge spirometers. Measurements included forced vital capacity (FVC), forced expiratory volume in one second (FEV<sub>1</sub>), forced expiratory flow at 75% of FVC (FEF<sub>75</sub>), 50% of FVC (FEF<sub>50</sub>), peak expiratory flow rate (PEFR) and inspiratory capacity (IC). Tests were administered according to standard procedures (36). At each test session, at least 2 paired maximum expiratory manoeuvres were performed by the subjects, until 2 spiograms were obtained that were reproducible to

within 5% for FVC and  $FEV_1$ . The spirometer was calibrated before and after each session for volume (using a 4 litre syringe) and flow (using a 10 L/sec rotameter).

Symptoms - Immediately after training runs, runners were asked to subjectively rate (using a modified Borg 0-10 scale) a number of respiratory and other symptoms, and also to rate the air pollution on the running route (Appendix II).

Questionnaires - Questionnaires evaluated individual training patterns (intensity, duration, frequency, mode), performance (training times, race times, self evaluation) and symptoms (breathing and leg fatigue rating) and any cold or allergy symptoms (Appendix III). Information on health was collected using the California Medical Survey, a personal data questionnaire and the American Thoracic Society Questionnaire (37).

#### 4) Statistical Methods

Arithmetic means, standard deviations, ranges and frequency distributions were employed for the statistical summaries of the variables which included the pollutant, symptom and pulmonary function variables and carboxyhaemoglobin.

The relationships between the environmental variables such as  $NO_2$  and temperature, and the response variables, which included the symptom variables, the pulmonary function variables and carboxyhaemoglobin were investigated using

multiple regression methods.

Backward stepwise linear regression was employed to assist in the identification of those combinations of environmental variables that best predicted the response variables. All regression models included as a factor each runner's ID code. Therefore, all relationships identified were between days within runners, i.e. taking into account events within individuals. The backward stepwise approach begins with a regression model that includes all of the environmental variables in the list of predictor variables. The predictor variable whose regression coefficient has the largest p value is deleted from the list of predictor variables, and a multiple regression analysis is repeated on the reduced list of predictor variables. Again, the predictor variable with the largest p value is deleted from the list, and once more a multiple regression analysis is performed on the reduced list. This process is continued until one has a regression model that can best predict the response variable. It is not correct to delete variables until all of the remaining variables have statistically significant regression coefficients, that is, have p values below some arbitrary level such as 0.05. The p value is a function of sample size, thus this approach does not guarantee the best model.

In trying to choose the best model, we must strike a balance between bias and precision. If we have too many

variables in our model, it will have low bias but high variance. If we have too few variables in our model, we will have low variance but large bias. One approach that is suggested and used by many researchers is to choose the model that has the lowest Mallows's CP statistic (38). The CP statistic measures the bias and variance of a regression model. However, because the CP statistic is based on a sample of data, it too can fluctuate. Therefore, this approach is simply a guide in picking out the best model.

Suppose that the final or "best" regression model identified by the stepwise procedure has three environmental variables. For example, as will be reported in detail in the RESULTS section, the regression model for the response variable POLLUTE, the subjective reporting of pollutant exposure, that is identified by the stepwise procedure, includes the three environmental variables  $\text{NO}_2$ , mean CO and average temperature. This model is not necessarily the model with the highest multiple correlation coefficient ( $R^2$ ) or the lowest Mallows's CP statistic among regression models that have three predictor variables. Furthermore, even if this were the case, and the stepwise procedure was successful in identifying the model with the highest  $R^2$  and the lowest Mallows's CP statistic, there may be other three variable models with only slightly smaller  $R^2$ , which make as much or more biological sense. This possibility can be tested using what is called "all possible regressions" analysis. After

identifying the number of variables that the model might have (backward stepwise regression), one performs a regression analysis on all possible combinations of three variables selected from the original list of variables. One then ranks these three variable models by their  $R^2$ . Thus one could then identify other models that would have been missed by the stepwise procedure, but are biologically meaningful and only slightly less statistically powerful.

The final analysis was multiple linear regression analysis, to obtain the p values associated with the regression coefficients in the competing models that were identified by the all possible regressions procedure. The models identified by the original screen of the stepwise regression analysis may include regression coefficients whose p values are larger than 0.05. This usually suggests that other models with similar predictive power may not include this variable. We suggest that for this reason, as well as because we are making so many comparisons, that only regression coefficients with p values less than 0.01 should be considered important by themselves. Furthermore, it has been our experience that only with such low p values will the variable sustain the more robust statistical analysis described below.

For significant relationships identified by the regression analysis, an analysis of individual slopes was carried out. For each individual runner, a linear regression slope was



calculated between the response and predictor variables identified by the regression analyses. The frequency distributions of these slopes were tabulated and their arithmetic means determined. The hypothesis that the mean of such a distribution is zero was tested with the one sample t test, the Wilcoxon signed rank test and the sign test. The latter tests are more appropriate for distributions that are skewed and have other departures from the Gaussian assumption. The slopes analysis is a more robust test of the relationships between exposure and response variables. It is not affected by the possible serial correlation between observations taken over time, as is the multiple linear regression analysis. Therefore, relationships found to be statistically significant by the regression analysis are sometimes not found to be significant in the slopes analysis.

## RESULTS

### 1) Subject Testing

Health testing of the runners was completed October 26, 1988. Over the 3 year study, 81 runners (50% of the club membership) were tested at least once, giving a total of 740 test-sets (sum of tests for all runners). Table 1A shows a breakdown of the number of repeat health tests on the runners. Since we examined the regression of pollution concentration vs. health variables (symptoms, pulmonary

function, and COHb), data could be used from runners who completed two or more repeat health tests, providing the pollution data on the corresponding health test days was also complete. Thus the final data set contained 70 runners (54 male, 16 female) and 712 test-sets, over 75 weeks of testing (Table 1B), completed over a "half" season (1986) and two complete run-testing seasons (1987 & 1988). Table 1C shows the summary of run distance and number of runners. The run distance ranged from 2 to 14 miles (3 - 22.5 km), although there were three main groups, 6 miles or 10 km (52 runners & 204 test-sets), 8 miles or 13 km (46 runners & 149 test-sets) and 10 miles or 16 km (58 runners & 337 test-sets). It should be noted that runners did not always run the same distance each week.

## 2) Pollutant Concentrations

Tables 2A-C show mean pollutant concentration over 1987 and 1988, for (a) the Gage mobile samplers (6 and 10 miles), (b) the Gage-Breadalbane site stationary samplers, and (c) the two Ontario Ministry of Environment (MOE) monitoring stations, one at 26 Breadalbane St., Toronto (MOE-Breadalbane) and the other at Evans/Arnold Ave., Etobicoke (MOE-Evans). Pollution data was reported for NO<sub>2</sub>, SO<sub>2</sub>, CO and particulates only if data was complete for these four pollutants at all stationary and mobile sites described above. A total of 34 "weeks" of complete Wednesday (runday)

pollution data was collected. There were only two days with complete data in 1986, thus they were not included in these Tables.

Table 2A (1987) shows that for  $\text{NO}_2$ : (a) the two MOE sites showed different levels, the Evans site being consistently higher (39 ppb) than the Breadalbane site (27 ppb), (b) the Gage-Breadalbane (30 ppb) and MOE-Breadalbane (27 ppb) were within 3 ppb, (c) overall there was no difference between Gage mobile (average of 6- and 10-mile) and MOE (average of Breadalbane and Evans) stationary sites, and (d) the maximum levels recorded ranged from 50 to 55 ppb for the five "sites", these levels are  $1/4$  of the 1-hr Ontario MOE Ambient Air Quality Criterion (AAQC) of 213 ppb and  $1/5$  of the Ontario MOE Point of Impingement (POI) standard of 267 ppb (limiting effect based on health).

Mean sulphur dioxide levels were all below 10 ppb. Maximum values were 30 ppb or lower, well below the 1-hr AAQC of 264 ppb and the 0.5-hr POI standard of 317 ppb. However, it should be noted that the limiting effect for  $\text{SO}_2$  takes into account both health and vegetation effects.

For carbon monoxide: (a) mean levels at the two MOE stations were similar for Breadalbane (1.1 ppm) and Evans (1.4 ppm), (b) the Gage 6-mile CO (4.3 ppm) was higher than the Gage-10 mile CO (2.7 ppm), (c) the two MOE stationary sites underestimated the CO exposure of the runners by 48% to 74% and (d) the maximum mean CO concentrations for the Gage

6-mile (7.4 ppm) and 10-mile (7.2 ppm) mobile monitors were approximately 1/4 of the 1-hr AAQC (31.6 ppm) and above the 0.5-hr POI standard (5.2 ppm), limiting effects based on health.

Respirable suspended particulate (RSP) matter showed that (a) the Gage 6-mile (104 micrograms/m<sup>3</sup>) was higher than the Gage 10-mile (72 micrograms/m<sup>3</sup>), and (b) the Gage-Breadalbane (55 micrograms/m<sup>3</sup>) underestimated the RSP exposure of the runners by 47% (Gage 6-mile) and 24% (Gage 10-mile). No comparisons can be made between the MOE total suspended particulate (SP) matter (Hi-Vol sampler, 24-hr average) and the Gage RSP measurements, due to different sampling methods, fractions collected, and sampling period. There is no AAQC or POI standard for non-specific RSP. Furthermore, for total SP, the limiting effects for the AAQC and POI standard are based on visibility, not health.

Table 2B (1988) showed similar trends for NO<sub>2</sub> and SO<sub>2</sub>, compared to 1987, although the maximum level at the MOE-Evans site was higher for NO<sub>2</sub> (100 ppb) and SO<sub>2</sub> (60 ppb). Carbon monoxide levels in 1988 also showed similar patterns as in 1987, although levels for the mobile samplers were approximately 40% lower than 1987 levels. This was possibly a result of changes in the 1988 running route, detailed in the March 1989 progress report (Appendix VIII). Again, as in 1987, the MOE stationary sites underestimated the runners CO exposure (Gage mobile samplers) by 35% to 73%. The RSP in

1988 for the Gage 6- and 10-mile mobile samplers was 34% and 24% lower than 1987 levels, respectively, following a similar pattern to CO. As in 1987, the Gage-Breadalbane sampler underestimated the runners RSP exposure by 52% (6-mile) and 40% (10-mile).

Table 2C shows combined 1987 and 1988 data. Differences between mobile 6-mile and 10-mile are most apparent for CO, RSP and to a lesser extent NO<sub>2</sub>, in all cases, the 6-mile level being higher. The MOE levels for CO, and Gage-Breadalbane for RSP (stationary, fixed-sites), underestimate the runners exposure (mobile levels) by up to 73%. Finally, the Gage-Breadalbane and MOE-Breadalbane levels were within 1 ppb, for both NO<sub>2</sub> and SO<sub>2</sub>, indicating that the two sampling methods (impingers vs monitors) were highly comparable.

Tables 3A-3C show mean NO<sub>2</sub>, SO<sub>2</sub> and CO concentrations for the same monitoring sites as Tables 2A-C, including all available data over the 75 weeks of pollution monitoring. In addition, the 1800 hrs. and 1900 hrs. averages are shown for the MOE sites. This is the data that was used to develop the regression equations relating pollutant concentration to pulmonary function, COHb and symptom scores. Comparing NO<sub>2</sub> concentrations (Tables 2C and 3A), the mean levels using data collected over all possible weeks were almost identical to the subset of 34 weeks in which complete data across all five monitoring sites was available. Also of note is the fact

that the 1800 hrs. and 1900 hrs. averages for the two MOE sites differed by only 1 to 2 ppb, suggesting a fairly constant  $\text{NO}_2$  level over this period. The same observations can be made for  $\text{SO}_2$  (Table 3B). Table 3C shows that the CO concentrations are slightly higher when all possible weeks were included in the means, compared to the 34 week subset (Table 2C). This may be explained by the higher CO concentrations measured in 1986, data that was not included in Table 2C. Thus in summary, the observations regarding Tables 2A-C can be considered representative of the complete data set that was used to develop the regression equations.

Tables 3D and 3E show mean CO concentration at specific sites along the running route measured using mobile, continuous reading electrochemical CO monitors, for the 6 mile (Table 3D) and 10 mile (Table 3E) routes. There was only one mobile CO monitor in 1986, which followed the 10 mile runners, thus Table 3D shows means for 1987 and 1988 only. The number of weeks with measurements at specific sites are not identical (a) due to missing data, (b) on occasion, runners altered their running route, thus adding or missing some sites (c) four new sites were added in 1987 (Tip Top Taylors, War Plane, four mile turn point and Budapest Park) after discussions at the March 6, 1987 MOE-Gage meeting, (d) due to Sky Dome construction in 1988, the Spadina bridge was closed to pedestrians, therefore runners bypassed this area and followed Front St. to Bathurst St.,

thus two new sites were added (Front and Portland, and Bathurst Bridge), and (e) in 1988, runners tended to bypass the Peter and Front site, cutting across at Peter and Wellington, thus this site was also added in 1988.

Table 3D shows that with two exceptions (Lakeshore and Bathurst, and Remembrance Drive-3 mile turn point), CO at each site along the route is higher on the way out than on the way back. These differences can be explained by the vehicular traffic burden, with peak traffic occurring during the "out" CO recordings, while during "back" recordings the "rush hour" traffic has decreased. Although the difference is only 0.9 ppm, the mean CO, all sites averaged, on the way out (3.8 ppm) is higher than on the way back (2.9 ppm). The peak or highest CO concentration on the way out (12.1 ppm) is also higher than on the way back (8.3 ppm). The mean CO concentration for the 6 mile route, all sites out and back, was 3.4 ppm over 54 weeks, similar to the 3.8 ppm mean in Table 3C for the Gage-6 mile CO monitor over 72 weeks, when the 1986 values were calculated using estimated values, as detailed in the Table 3C footnote.

Table 3E, with data from the 10 mile bicycle, shows a similar trend to the 6 mile bicycle, that is, with three exceptions (Front and Portland, Canada Malting, and the War Plane), the CO concentration at each site along the route is higher on the way out than on the way back. The same pattern is seen for peak and mean CO levels, the "out" value being

higher than the "back" value. The mean CO concentration for the 10 mile route, all sites out and back, was 2.7 ppm, lower than the 3.4 ppm recorded for the 6 mile route.

Table 4A compares mean temperature for Wednesday runs over 1986 to 1988, for the Gage 10 mile bicycle, the Evans Ave. MOE site and the Trinity College Atmospheric Environment Services (AES) site. The mean temperature was 17 - 18°C for the three sites, suggesting that any site could be used to estimate the ambient temperature. Furthermore, the temperature range for the three sites was consistent across the sites, with a low of 2°C and a high of 31 - 33°C. The reading of 5°C for the Gage 10 mile bicycle (low range), compared to 2°C for the other sites, is due to the fact that temperature was not recorded (Gage - 10 mile) on the coldest day (Nov. 11, 1987). The Evans MOE site showed that there was no change in temperature from 1800 hrs. to 1900 hrs., indicating a similar thermal load for the 6 and 10 mile runners.

Mean relative humidity (RH) is reported in Table 4B for the Gage 10 mile bicycle and the Trinity College AES site, over 1987 and 1988. No Gage RH was collected in 1986, although there was AES data. The mean RH for the Gage monitor (60%) was very similar to the AES site (58%), as were the ranges, again suggesting that either site could be used to estimate the runners humidity exposure.



### 3) Health Effects

Table 5A shows subjective ratings of 8 symptoms and a subjective rating of the pollution during Wednesday runs over 1986 - 1988, for all run groups combined. Runners were asked to rate the 8 symptoms and pollution exposure immediately after their run, using a modified Borg scale ranging from 0 (symptom was not noticeable or no pollution) to 10 (very, very strong, almost max symptom severity or pollution). The POLLUTION RATING, LEG FATIGUE AND BREATHING FATIGUE were recorded during the entire study, while the other 6 symptoms were added the third week of 1987 (after reviewing the 1986 data, and from experience with previous environmental chamber studies using these symptoms). Due to time constraints, the symptoms and pollution rating were not always obtained. This is reflected by the number of test-sets (N) which ranged from 464 to 700, out of a total of 712 (Table 1B). The highest ratings were shown for the average POLLUTION RATING (3.3), LEG FATIGUE (3.3) and BREATHING FATIGUE (2.9), which were in the moderate range (Borg rating of 3), although they varied from a low of 0 to a high of 10. For the POLLUTION RATING, in 69% of the test-sets, a rating of 3 or greater was recorded, while the corresponding values for LEG FATIGUE and BREATHING FATIGUE were 67% and 61%, respectively. Lower mean ratings were reported for respiratory and nasal symptoms and EYE IRRITATION; these were in the very weak (1) to weak (2) range. Among these six symptoms, only NASAL DISCHARGE and

SPUTUM/PHLEGM had a maximum rating of 10 reported, while the remaining four symptoms had maximum ratings of 7 or 8. Approximately 70% of the reports were trace (0.5) or higher for NASAL DISCHARGE and STUFFINESS, and SPUTUM/PHLEGM, while the corresponding values were 60% for EYE IRRITATION, 54% for SORE/DRY THROAT and 38% for COUGH.

Table 5B shows the average symptom and pollution ratings from Table 5A, categorized into the three main run groups, 6, 8 and 10 miles. The ratings appear to be independent of the run distance and show no consistent trends.

Results of background anthropometric and pulmonary function test results performed at The Gage Research Institute are shown in Table 6A. Runners may have performed up to three of these tests, however, only the initial test results are shown in this Table. The initial background tests were performed only after runners had completed at least four Wednesday run tests, and remained actively involved in the study. The background tests were completed immediately prior to (on the same day) the treadmill tests. Background data was completed on 45 of the 58 runners in the study who completed four or more Wednesday run tests, and included 34 males and 11 females. The 13 who did not have background testing, either dropped out of the study, or had declined when asked to do the tests. The average age of the runners was 37 yrs, and ranged from 17 to 57 yrs, the male

runners being slightly older (38 yrs) than the female runners (34 yrs). The runners were of average height for their gender, while as expected, they were lean, the mean weight being 69 kg for males and 55 kg for females. The background flow-volume data was performed on an Eagle spirometer (water filled 8 liter bell) with microprocessor (Collins Eagle One). All values were in the normal range of 80 - 120% of predicted normal values for FVC and FEV<sub>1</sub>, and 50 - 150% for FEF<sub>50</sub> and FEF<sub>75</sub>; in fact, the mean FVC, FEV<sub>1</sub> and FEF<sub>50</sub> were above 100% of the predicted normal values (39 - 41), but still within the normal range. The percent of predicted normal values of FEF<sub>50</sub> and FEF<sub>75</sub> were slightly higher for females than males. The mean lung volumes, airway resistance and single breath CO diffusion capacity of the lung were all within the normal limits. These data indicate that the runners were very healthy in terms of their respiratory function.

Table 6B shows results of the pulmonary function and COHb% tests performed immediately before the Wednesday runs, over 1986 - 1988; this data was used as the reference or baseline measurements for Tables 6C-E, where changes in pulmonary function over the run were calculated. The flow-volume measurements were made using a Wedge Spirometer and X-Y recorder. The flow-volume variables were hand calculated from the X-Y tracing. Comparing Table 6B with 6A, and keeping in mind that the ratio of males to females was different (~4:1 in Table 6B and ~3:1 in Table 6A), the

variables FVC, FEV<sub>1</sub>, PEF<sub>R</sub> and FEF<sub>50</sub> were within 5% of Table 6A, indicating that the hand calculated values were comparable to the microprocessor calculated values. The higher FEF<sub>75</sub> in Table 6B compared to 6A can be explained by the larger percentage of males in Table 6B, males having a larger FEF<sub>75</sub> than females.

The two-minute oxygen rebreath estimate of blood carboxyhaemoglobin percent shows a mean prerun value of 0.4%. Baseline COHb% in non-smokers is 0.4%, due to endogenous CO production, but may be 1.0 to 1.5% due to urban air pollution, such as car exhaust, or exposure to "second hand" cigarette smoke. Smokers, on the other hand, have an elevated COHb% compared to non-smokers, primarily dependent on the amount smoked. There were only three runners with a prerun COHb% above 1.5%, one of which was a smoker of 15 cigarettes per day; we could not confirm if the other two runners smoked. Thus the two runners may have been current smokers, or alternatively, had a very high CO exposure prior to the Wednesday prerun testing. There were two additional runners who had a prerun COHb% above 1.0%, neither of them were smokers; we assume exposure was due to urban CO exposure and/or "second hand" smoke exposure at work before the testing. Among 21 runners who answered respiratory health questionnaires, 14 had never smoked, 16 were ex-smokers and one was a current smoker.

Table 6C shows the mean pulmonary function and COHb%

absolute change (post run minus prerun), over 1986 - 1988. Over the run, mean pulmonary function changes were small, with decreases in FVC, IC, PEF<sub>R</sub>, and FEF<sub>50</sub>. There were small increases in FEV<sub>1</sub> and FEF<sub>75</sub>. For most of the pulmonary function variables, the maximum increase and decrease were fairly similar in magnitude about the zero or no change value. However, the COHb% (mean absolute decrease of 0.07%) ranged from an absolute decrease of 1.54% to an absolute increase of 0.26%. Only 8 or 1.2% of the test-sets (5 runners) showed an increase in COHb% over the run.

Table 6D shows the same variables as Table 6C, expressed as a percent change, that is, the absolute change/prerun value x 100. The mean changes in pulmonary function were small, less than 5%, although the ranges showed a greater variability, with substantial increases and decreases. If a change of 15% is taken as a reference point, for FVC, FEV<sub>1</sub>, IC and PEF<sub>R</sub>, more runners (test-sets) showed decrements of 15% or greater over the run than increases of 15% or greater. The absolute number of decreases and increases, of 15% or greater were as follows: for FVC 1 and 0, FEV<sub>1</sub> 2 and 1, IC 61 and 7, and PEF<sub>R</sub> 6 and 4, respectively. For FEF<sub>50</sub> and FEF<sub>75</sub>, a different pattern was seen, fewer runners showed a decrease of 15% or greater, than an increase of 15% or greater, the absolute numbers being 35 and 56 for FEF<sub>50</sub> and 75 and 137 for FEF<sub>75</sub>.

Table 6E compares the three run groups, 6, 8 and 10

miles, for the variables in Table 6D, that is percent change in pulmonary function and COHb%. There were no dramatic differences between the three run groups.

#### 4. Exposure Response Relationships

The frequency distributions for the six symptom variables, SORE/DRY THROAT, COUGH, SPUTUM/PHLEGM, STUFFY NOSE, NASAL DISCHARGE and EYE IRRITATION are given in Table 7, and supplement the information already presented in Table 5A. The rating scale used was a 10 point severity scale. One can see that on 28% to 62% of the days, runners actually reported experiencing absolutely no trace of symptoms (rating of 0). The frequency distributions for BREATHING FATIGUE, LEG FATIGUE and POLLUTION RATING are given in Table 8. For these three variables and particularly for the Pollution Rating scale, there were very few days on which runners reported no trace of symptom. In fact, on more than 60% of the days, the runners reported at least a moderate level (rating of 3).

The results of the stepwise regression analysis for the exposure and symptom variables shown in Table 9 utilize the pollutant concentrations measured by the Gage personal monitors that were placed on the bicycles that accompanied the runners on their route. Since the runners did not run only 6 and 10 miles, that is, the same routes covered by the 6 and 10 mile mobile Gage samplers, the Gage pollutant concentrations used in the regression equations were as

follows: (a) if the distance run was less than or equal to 6 miles, the 6 mile concentration was used, (b) if the distance run was greater than or equal to 10 miles, the 10 mile concentration was used and (c) if the distance run was less than 10 miles or greater than 6 miles, the mean of the 6 and 10 mile concentrations was used. From inspection of Table 9 we see that the response variable SORE/DRY THROAT is related positively to the variable pollution rating (POLLUTE) and negatively to the variable average relative humidity (AVRH). The regression coefficient for POLLUTE is 0.18 with a p value of 0.001, that is, the positive relationship between SORE/DRY THROAT and POLLUTE is strongly significant. The negative regression coefficient of 0.014 for AVRH, although statistically significant ( $p=0.002$ ), indicates that for a change of relative humidity of 1% there will be a change of only 0.014 in the severity score for the variable SORE/DRY THROAT.

The most consistent predictive variable was POLLUTE, which measured the runner's subjective feeling about the level of pollution experienced during the run. In all cases, the sign of the partial regression coefficient was positive, indicating that on days the runner reported a higher pollution rating, the runner also reported a higher symptom rating. For the three respiratory symptoms SORE/DRY THROAT, COUGH and SPUTUM/PHLEGM and for the symptom NASAL DISCHARGE, a negative relationship was found with average relative

humidity. A negative relationship was found between average temperature (AVTEMP) and the symptoms SPUTUM/PHLEGM, STUFFY NOSE and NASAL DISCHARGE. The pollutant  $\text{SO}_2$  showed only weak positive relationships with STUFFY NOSE ( $p=0.04$ ) and SPUTUM/PHLEGM ( $p=0.06$ ).

The consistent relationships found for the pollution rating scale POLLUTE, suggested that it would be interesting to discover what exposure variables would be significant if it were left out of the model. Furthermore, the variable POLLUTE could be included as a response variable in its own right. The results of these analyses that excluded POLLUTE from the list of predictor variables are given in Table 10, as summarized below.

For the symptom variables SORE/DRY THROAT, COUGH, SPUTUM/PHLEGM, STUFFY NOSE and NASAL DISCHARGE, the relationships with AVRH and AVTEMP were very similar to what was shown in Table 9. The weak ( $p=0.04$ ) relationship between STUFFY NOSE and  $\text{SO}_2$  shown in Table 9 was replaced in Table 10 by a similarly weak ( $p=0.06$ ) relationship with  $\text{NO}_2$  and a similar ( $p=0.04$ ) relationship with PEAKCO. The variables BREATHING FATIGUE and LEG FATIGUE which were related to the variable POLLUTE, are shown to be strongly related to temperature, which is itself strongly positively related to the pollution rating scale POLLUTE.

The pollution rating scale (POLLUTE), as a response variable, is shown to be strongly related to  $\text{NO}_2$  ( $p=0.001$ ),



mean carbon monoxide ( $p=0.002$ ) and mean average temperature ( $p=0.001$ ).

The results of similar analyses for the pulmonary function response variables are given in Tables 11 and 12. The results for the analyses that include the predictor variable POLLUTE are given in Table 11, and those excluding POLLUTE given in Table 12. Inspection of these two tables suggest that only the negative relationship between  $\text{NO}_2$  and  $\text{FEF}_{75}$  measured at the end of the run ( $\text{FEF}_{75}\text{POST}$ ) is both statistically strong ( $p < 0.005$ ) and biologically meaningful, that is, when  $\text{NO}_2$  concentration is high,  $\text{FEF}_{75}\text{POST}$  is low.

The structure of Tables 13, 14, 15 and 16 is parallel to those in tables 9, 10, 11 and 12, except that the pollutant measures made by the Ontario Ministry of the Environment (MOE) at their Bredalbane station are included in the list of predictor variables, along with those measured with the Gage portable samplers. Pollutant concentrations used at the MOE Bredalbane site were the average of the 1800 hrs. and 1900 hrs. mean values (see Table 2A) for the gaseous pollutants, coefficient of haze (BCOH) and the Air Pollution Index (BAPI), and 24-hr. averages for particulates (BPART), sulphates ( $\text{BSO}_4$ ), and nitrates ( $\text{BNO}_3$ ), collected on Wednesday run days. The gases included CO (BCO), hydrocarbons using x-ray analysis (BHGX), NO (BNO),  $\text{NO}_2$  ( $\text{BNO}_2$ ),  $\text{O}_3$  ( $\text{BO}_3$ ), and  $\text{SO}_2$  ( $\text{BSO}_2$ ). Because of the extra days on which MOE data were not collected, the sample size has dropped from  $N=423$  in Table 9

to N=335 in Table 13. Many of the relationships found with POLLUTE, AVRH and AVTEMP and given in Table 9, were again found in Table 13, but often with a larger p value, that is, less statistically significant. This is not surprising, because of the smaller number of observations in the datasets. For example, in Table 9, the regression coefficient between SORE/DRY THROAT and POLLUTE is 0.18 with a p value of 0.001, whereas in Table 13 the regression coefficient is 0.16 with a p value of 0.04.

Some relationships exist in Tables 13 and 14 for which explanations are not readily apparent. For example, hydrocarbons (BHCX) are negatively related to SPUTUM/PHLEGM and LEG FATIGUE. The intercorrelations between hydrocarbons and other pollutants have not yet been extensively studied in an attempt to explain such anomalies.

Table 14 shows that the three Gage predictor variables, average temperature (AVTEMP), NO<sub>2</sub> and mean carbon monoxide (MEANCO), that were originally found to be positively related to the pollution rating scale POLLUTE (Table 10), were again found to be the most important predictor variables of POLLUTE.

The results of the analyses of the pulmonary function variables are given in Tables 15 and 16. The relationship found previously between FEF<sub>75</sub>POST and the Gage measured NO<sub>2</sub> is again found in Tables 15 and 16. In addition are found a negative relationship between FEF<sub>75</sub>POST and the MOE measured

SO<sub>2</sub> and a positive relationship with the MOE measured SO<sub>4</sub> (BSO<sub>4</sub>). The positive relationship found between FEF<sub>75</sub>POST and BSO<sub>4</sub> and the positive relationship between FVCPOST and MOE measured NO<sub>3</sub> are not easy to explain. As for the case of hydrocarbons and SPUTUM/PHLEGM, mentioned above, such apparently surprising findings may be due to the intercorrelations among the environmental variables.

#### MODELS FOR THE REBREATHE CARBON MONOXIDE

The rebreathe estimate of the carboxyhaemoglobin (COHb%), measured pre and post run, is a biological marker for the amount of carbon monoxide (CO) that has actually entered and remained in the body. Tables 17 and 18 show the results of the stepwise regression analyses using the rebreathe estimate (RBCO) as the response variable. The analysis reported in Table 17 includes the pollution rating scale (POLLUTE) in the list of predictor variables, whereas the results reported in Table 18 do not. Not surprisingly, the mean carbon monoxide concentration obtained from the Gage portable samplers (MEANCO) is very strongly related to the RBCO. There is a suggestion of a negative relationship between RBCO and the peak concentration of CO (PEAKCO) over the run. This suggests that for a given CO concentration, the level of RBCO will be higher at the end of the run if the exposure was uniform over the run, rather than if it was received in a large pulse at some point during the run. This might simply

mean that in a run in which a large pulse or "pocket" of CO is experienced, there may be some time in which to exhale it from the body before completion of the run and/or lack of time to absorb it. The RBCO at the end of the run (RBCOPOST) is also strongly ( $p=0.0004$ ) related to the NO<sub>2</sub> concentration over the run.

#### MODELS FOR THE POLLUTION RATING SCALE

Because of the statistical significance found between the pollution rating scale (POLLUTE) and the exposure variables NO<sub>2</sub>, temperature and carbon monoxide, an all possible regressions analysis was conducted to determine whether other subsets of exposure variables would lead to models which had biological interest, but only slightly less statistical power. These analyses whose results are shown in Tables 19, 20 and 21 are based on the same 545 runner days of information for which complete information existed for the Gage variables NO<sub>2</sub>, average temperature, mean CO, mean RBCO and respirable particulates, and the MOE-Breadalbane ozone.

In Table 19 is reported the model which is almost identical to that reported at the bottom of Table 10. The analysis reported in Table 10 is based on 423 runner days, whereas the analysis reported in Table 19 is based on 545 runner days due to the increased number of missing observations for the individual symptoms which were not included in the analysis

reported in Table 19. It is reassuring that two analyses based on datasets that differ in size by more than 20 percent achieve such similar answers. For example, the regression coefficients for  $\text{NO}_2$ , MEANCO and AVTEMP from Table 10 are 0.009, 0.14 and 0.049, whereas from Table 19 they are 0.009, 0.13 and 0.049. Such consistency between models that result from using different datasets adds to the strength of our findings.

In Table 20 we see the slight loss in  $R^2$  from 0.414 (Table 19) to 0.409 that occurs when the mean (average of pre & post run) rebreath carbon monoxide variable (MEANRBCO) replaces mean carbon monoxide (MEANCO) in the model. Table 21 shows the model that includes respirable particulate concentration measured by the Gage portable sampler and ozone concentration measured by the Ministry of the Environment at their Breadalbane station. Again we see that this model is almost identical in statistical power to the other two models (Tables 19 and 20). It is not surprising that ozone might replace temperature in the model because of the high correlation of 0.65 (not reported in any Table) that we found between these two variables. However, the correlation coefficient between CO and respirable particulates was found to be only 0.14, and therefore its inclusion in the model has to do with the joint effect that it and ozone has on the variable POLLUTE.

A further regression analysis which is not reported here was performed between POLLUTE and the four variables CO, NO<sub>2</sub>, temperature and ozone. In this model, temperature remained statistically significant, while ozone did not. This suggests that part of the original significant relationship between ozone and POLLUTE was due to the high correlation between ozone and temperature, and was not only a direct relationship between ozone and POLLUTE.

#### SLOPES ANALYSIS

Because of the importance of the findings reported for the pollution rating scale, further analyses were conducted to determine if the relationships that were uncovered by multiple regression could be confirmed by another approach. This approach involved the calculation of a regression slope for each individual, relating the pollution rating scale (POLLUTE) and a particular air pollutant. If there were no relationship between the pollution rating scale and the air pollutant, then we would expect half of the slopes to be positive and half negative. However, if there is a positive relationship between the pollution rating scale and the air pollutant, then we would expect a preponderance of positive slopes. In other words, with a positive slope, when the air pollutant concentration is high, the subjective report of air pollution severity (POLLUTE) is also high.

In Table 22 we see that 69% of the slopes between POLLUTE

and mean  $\text{NO}_2$  are positive, which is significantly ( $p=0.004$ ) more than the 1:1 split anticipated from the null hypothesis. The mean slope is 0.021, which means that for each unit ( $\text{microgram}/\text{m}^3$ ) change in  $\text{NO}_2$  there would be expected a 0.021 change in the POLLUTE variable. The median slope is 0.011. These values should be compared to the regression coefficient of 0.009 reported in Table 19. Similar to the findings of Table 22, 68% of the slopes for POLLUTE against mean CO (Table 23) were positive, with a mean slope of 0.10 and a median slope of 0.075. For the relationship between POLLUTE and temperature, shown in Table 24, the mean slope was 0.058 and the median slope was 0.044. Summarizing Tables 22-24, the slopes relating the subjective report of pollution rating with  $\text{NO}_2$ , CO and temperature, were positive in 68-69% of the runners, and the p values for the null hypothesis that the slopes were zero, ranged from 0.004 to 0.006.

The mean slope for the relationship between rebreathed CO measured at the end of the run (RBCOPOST) and mean CO is 0.88 and the median slope is 0.71 (Table 25). Eighty-four percent (48/57) of the runners had a positive slope, and the p value for the sign test was highly significant ( $p < 0.0001$ ). The mean slope for the relationship between RBCOPOST and mean  $\text{NO}_2$  is 0.031, and the median is 0.033 (Table 26). Again, as shown in Table 25, a high proportion of runners had positive slopes (74%), and the p value for the sign test was 0.0006.

## COMPARISON OF PORTABLE SAMPLERS WITH MOE SAMPLERS

The important relationship between POLLUTE and the three variables  $\text{NO}_2$ , CO and temperature was uncovered by regression analysis and confirmed by a slopes analysis. The effect of replacing the estimates of CO and  $\text{NO}_2$  obtained by using the Gage portable samplers by the corresponding estimates obtained from the Breadalbane MOE station was investigated. In turn, one or another or both of the portable sampler estimates of CO and  $\text{NO}_2$  were replaced in the multiple regression analysis by their MOE counterparts, and the results given in Table 27. The regression coefficient between the Gage CO and POLLUTE is 0.1235, with a p value of 0.0071 (Model A). When the CO estimate obtained from the Gage portable sampler was replaced by the MOE-Breadalbane estimate, the regression coefficient was -0.084, with a p value of 0.31 (Model C). Therefore, the statistically significant positive relationship between Gage CO and POLLUTE became negative and nonsignificant when the Gage CO was replaced by the Breadalbane - CO. In Model A, the significant positive relationship between POLLUTE and the Gage  $\text{NO}_2$  ( $p=0.0017$ ) was weakened by the replacement of the portable sample estimate of  $\text{NO}_2$  by the MOE estimate ( $p=0.52$ ) in Model B. Interestingly, this weakening was less ( $p=0.021$ ) if the MOE estimate of CO was allowed to remain in the model (Model D).



### Discussion

On the majority of the 75 training runs, recorded pollutant levels including  $\text{NO}_2$ ,  $\text{SO}_2$  and CO were reasonably low, with Gage and MOE-Breadalbane maxima being 17-41% of the 1-hr average Ontario Ambient Air Quality Criteria. The MOE-Evans site showed higher maximum levels for these three pollutants, but due to a preponderance of missing values, this data was not used in the regression analyses. The concentrations measured using our sampling systems and those used by the MOE, both at the Breadalbane site, were the same, giving us confidence in our data from the Gage mobile samplers. The results show that CO and RSP at the fixed MOE sites (Breadalbane and Evans) are lower than the mobile "personal" samplers, and that the 6 mile mobile sampler concentrations are higher than the 10 mile concentrations. Therefore, using the MOE values would underestimate the runners "personal" exposure. Also, the 6 mile and 10 mile runners are exposed to different concentrations. These findings illustrate the importance of using "personal" sampling when assessing exposure of mobile subjects. A possible explanation for the difference in 6 and 10 mile levels is that the 10 mile route extended out along the Martin-Goodman bicycle/jogging trail, between Lake Ontario and Lakeshore Boulevard. Traffic is lighter and less congested there compared to the downtown core, also it is more open (parkland with few buildings) and on-shore breezes

may "clean" the area. Furthermore, the 10 mile group returns to the downtown core approximately 1/2 hour later than the 6 mile group, when rush hour traffic is reduced and pollution concentrations are lower, as suggested by lower CO concentrations that were recorded on the return route. Therefore, the "6 mile runners", who spend more of their time in the downtown core compared to the 10 mile group, may be exposed to higher pollution concentrations, although the actual pollutant dose or burden may be similar for the 2 groups, as the 10 mile group is exposed for a longer time.

The percentage of days on which runners reported symptoms whose severity was at least moderate (rating of 3) was 19% for sore/dry throat, 12% for cough, 36% for sputum/phlegm, 33% for stuffy nose, 37% for nasal discharge and 17% for eye irritation. With the exception of eye irritation, the frequency of a reported severity rating of moderate or greater, for these five symptoms, was similar or higher in this study of runners, compared to a chamber study (35), in which 24 healthy nonsmokers were exposed to a "moderate" level of "second hand" cigarette smoke (mean level of 17 ppm CO). Because the present study population consisted of a group of runners who were questioned about their symptoms immediately after their run, it is not surprising that there was a high percentage of days on which at least a moderate level of breathing fatigue and leg fatigue was reported, the

respective values being 61% and 67%.

The relationship observed between the symptom variables and the subjective rating of pollution were the strongest and most significant. Furthermore, it was found that the subjective rating of pollution was jointly related to breathing fatigue, eye irritation and sore/dry throat, suggesting that the subjects' symptom reporting was related to their perceptions of the pollution levels they were exposed to.

Statistical methods could have been employed to derive a symptom complex comprised of some of the 8 symptoms. The reasoning for such a symptom complex is that exposure to pollutant(s) among a group of individuals may provoke varied responses. That is, pollution may affect the eye of one individual, but in another individual may affect the nose or breathing. To the extent that this supposition is true, it may explain why the relationships observed between the exposure variables and the individual symptom variables are neither consistent nor all that strong, with the exception of temperature and humidity. The construction of such a symptom complex would have to take account of the fact that a variable such as temperature had a positive relationship with some symptom variables and a negative relationship with others. A stepwise multiple linear regression between the

pollution rating and the other 8 symptom variables (not reported), revealed a significant relationship with the symptoms breathing fatigue, eye irritation and dry/sore throat. That is, the pollution rating scale may be an integrator of many different symptoms. Therefore, it seems that the pollution rating scale achieves that which a symptom complex is intended to do. For example, the effect of carbon monoxide, which shows up weakly for the individual symptoms cough, stuffy nose and eye irritation is much stronger with the pollution rating scale. The pollutant  $\text{NO}_2$  is only weakly related to stuffy nose and eye irritation, but is more strongly related to the pollution rating scale. The positive relationship between mean temperature and the pollution rating scale appears to capture the similar relationships between temperature and each of the symptoms breathing fatigue and leg fatigue.

The negative relationships of the symptoms with relative humidity and temperature are not surprising. Temperature (dry bulb) and relative humidity (RH) are interrelated and effects of RH are really temperature effects. Relative humidity is a measure of both dry and wet bulb temperature, and specifically, is the percentage of moisture in the air relative to the amount it could hold if saturated at the same temperature. Dry air (low absolute humidity) and low temperatures are irritants to the respiratory tract. If we

look at the respiratory and nasal symptoms as a response to irritation, we would expect to see throat irritation (sore/dry throat) increase as both RH and temperature decrease. Similarly, cough and sputum/phlegm as responses to irritation would increase as humidity and temperature decrease. However, cough also increases with very high humidity, as shown in studies with inhalation of hypotonic saline. In hospitals, patients are given humidified air to loosen their sputum. Sputum decreases and becomes thicker with hot dry air.

Cold increases nasal stuffiness because of vasodilation, and if it is hot, there is vasoconstriction and a decreased sensation of nasal stuffiness. Cole et al. (42) observed in resting subjects that there was an increase in nasal stuffiness with decreased temperature. However, they found that nasal stuffiness decreased with exercise, overriding the temperature effect. Nasal discharge probably reflects the same thing as nasal stuffiness, that is, the irritation effect of low temperature causes the nose to run.

The effects of relative humidity are really temperature effects, such that the body responds in order to lower temperature by evaporating water, as water has the highest latent heat of evaporation and this mechanism acts to keep core temperature stable, at the equilibrium or "set point". This mechanism helps explain the positive relationships between breathing fatigue and leg fatigue with temperature.

With respect to breathing fatigue, as temperature increases, the work of breathing increases. Leg fatigue is most likely a response to breathing fatigue and high temperature, related to lactic acid accumulation caused by an oxygen debt due to anaerobic respiration. Fatigue comes from diversion of blood flow to the skin.

Therefore, the findings of this study, related to symptom reports, can partially be explained by both the effects of exercise and the interactive effects of temperature and relative humidity.

The stepwise regression procedures identified many significant relationships that were not followed up in detail in this report. However, the negative relationship between the post run  $FEF_{75}$  and the Gage sampled  $NO_2$  was strongly statistically significant. This relationship was confirmed by the slopes analysis (not reported). However, a weakly significant positive relationship was found between the post run  $FEF_{75}$  and  $SO_2$  ( $p=0.04$ , Table 11). We have cautioned that a regression coefficient with a p value greater than 0.01 should not be taken too seriously. Furthermore, as shown in Table 12, when the analysis was repeated on a slightly different dataset, the p value associated with the regression coefficient for  $SO_2$  increased to 0.12, whereas the originally strong negative relationship with  $NO_2$  remained. This provided justification for our original position on data

interpretation. Therefore, although the negative relationship between  $\text{NO}_2$  and  $\text{FEF}_{75}$  appears to be real, it stands alone among the relationships between pulmonary function and pollutants that make biological sense. The post run  $\text{FEF}_{75}$  was also weakly related to temperature. This relationship was much weaker than that found between temperature and the symptom variables.

The finding that symptom severity was related to pollution concentration, but that, in general, measures of pulmonary function were not, is consistent with the findings of Dockery et al (43). In this study, increases in respiratory symptom reporting were associated with annual mean particulates, but no relationship between pulmonary function and pollution concentration was found. They concluded that air pollution exposure may increase respiratory symptom rates with no resulting permanent compromise in lung function.

The results of the present study suggest that symptom reporting may be a more sensitive or subtle indicator of effects of air pollutants than pulmonary function, and that the subjects' perception of pollution levels may be an integrator of the symptom complex that results from exposure.

The observed relationship between the subjective rating

of pollution and the local concentrations of  $\text{NO}_2$  and  $\text{CO}$ , suggests that the pollution rating is a reflection of, or is influenced by, local car exhaust emissions. While  $\text{NO}_2$  and  $\text{CO}$ , by themselves, may not cause the symptoms, they may be indicators of car exhaust emissions, and the actual irritant response may be to local car exhaust emissions that we are not measuring locally (with the personal samplers). Similarly, peak  $\text{CO}$  concentration may be an indicator of "pockets" of car exhaust emissions, and these would not likely be picked up by remote, fixed-site MOE monitoring. Therefore, the fact that relationships are strongest and most consistent with the mobile or "personal" concentrations, and variably, inconsistently, or not at all related to the levels measured at the fixed remote sites, emphasizes the importance of measuring exposure as close to the subjects as possible. This is consistent with previous studies of personal exposure. Similarly, Bates & Sizto (44-46) found that daily  $\text{SO}_4$  data from one central monitoring site were not correlated with respiratory admissions, but that using the data from 17 stations throughout the region were.

For each runner, the dose of air pollutant that was inhaled would be a product of the air pollutant concentration over the run, the ventilation rate during the run and the time taken to complete the run.

It was not possible to measure the ventilation rate during



the run, and instead, each runner measured his/her heart rate at the mid-point of the run. On a separate occasion, the log linear relationship between a runner's ventilation rate and heart rate was determined from measurements taken while the runner was on a treadmill. With these laboratory data, one could estimate ventilation rate from heart rate, during training runs. Therefore the DOSE is given by

$$\text{DOSE} = \text{POLLUTANT CONCENTRATION} \times \frac{\text{RUN (min)}}{\text{TIME}} \times \text{ESTIMATE OF (Litres/min) VENTILATION}$$

When the calculated dose of Gage NO<sub>2</sub>, CO, and respirable particulates, and MOE-Breadalbane ozone replaced the respective individual concentrations of Gage NO<sub>2</sub>, CO, respirable particulates, and MOE-Breadalbane ozone in the regression models with pollution rating, reported in Tables 19, 20 and 21, the level of statistical significance was lowered (the p value increased). Therefore, replacing concentration by dose in no instance improved the regression model for the response variable pollution rating. This could be due to the assessment of heart rate, which has its own error associated with it, and in fact, is also temperature sensitive. It could be due to the estimation of ventilation rate from a predictive model that was created with data far removed in time from many of the run days on which it was used. Furthermore, the predictive model does not take into account the influence of temperature, humidity or wind, factors that are highly variable in the outdoor environment, in comparison to the controlled laboratory conditions. The

run time itself is probably quite accurately measured, and probably is not responsible for the inability for dose to improve the regression models.

Rebreathe CO also provides an estimate of dose, that is, inhaled CO bound to the haemoglobin. However, when mean rebreathe CO replaces mean CO in the regression model with pollution rating as the response variable, not only does the  $R^2$  go down slightly, the p value drops from 0.004 to 0.046, i.e. not statistically significant by our criteria. One might think that this should not be the case, because the mean rebreathe CO estimates the concentration of CO in the runner's blood, and therefore should be a more accurate indicator of dose than the local environmental CO concentration measured by the Gage monitor placed on the bicycles. However, the air concentration of CO, because of its correlation with other air pollutants, includes the effect of other air pollutants, and therefore for that reason might be expected to be more strongly related to the pollution rating scale. The lack of resolution of mean rebreathe CO is not surprising, given that it includes the prerun rebreathe CO, and therefore depends on the subjects' exposures to CO previous to the run, such as during the day or in transit. The half-life for CO elimination may range from 2 hr to 6.5 hr in resting subjects. Thus the residual

effects of the prerun CO exposure will influence both the pre and post run rebreathe CO. However, such a comparison between the size of the effect of local CO and rebreathe CO would only be appropriate if one were certain that the two estimates of CO, in the blood and in the air, were equally reliable. Furthermore, much of this discussion is academic because as shown in Tables 19 and 20, the difference in the statistical power of the models is extremely small.

The finding that the pollution rating is related to ozone and particulate exposure is consistent with the findings of Bates and Sizto (44-46), who observed increased hospital admissions with ozone and  $SO_4$ , and Dockery et al (43), who observed increased symptomatology with particulates. The fact that the current study could not see consistent relationships with  $SO_4$  may relate to the fact that using one station to assess  $SO_4$  exposure was not appropriate, as shown by the Bates' study which used 17 stations through the region to uncover the relationships.

In summary, the results suggest that 1) symptoms and subjective rating of severity of pollution are related to pollution levels, 2) pulmonary function for the most part is not affected or minimally affected by the exposures encountered in this study, 3) the strongest relationships are seen using levels measured by the mobile personal samplers.

REFERENCES

1. McCafferty, W.B. Air pollution and athletic performance. Springfield, III: C.C. Thomas, 1981.
2. Folinsbee, L. Effects of air pollutants. In: Current Therapy in Sports Medicine. Ed. P. Welsh & R.J. Shephard. Burlington: B. Decker, 1985.
3. Shephard, R.J. Carbon monoxide - the silent killer. Springfield, III: C.C. Thomas, 1983.
4. Shephard, R.J. Athletic performance and air pollution. Can. Med. Assoc. J. 131:105-109, 1984.
5. Nicholson, J.P. & Case, D.P. Carboxyhemoglobin levels in New York City Runners. Phys. Sports. Med. 11:135-138, 1983.
6. Kjeldsen, K., Thomas, H.K. & Astrup, P. Effects of carbon monoxide on myocardium: ultrastructural changes in rabbits after moderate, chronic exposure. Circ. Res. 34:339-48, 1974.
7. Astrup, P., Kjeldsen, K. & Wanstrup, J. Enhancing influence of carbon monoxide on the development of atheromatosis in cholesterol-fed rabbits. J. Atheroscleros. Res. 7:343-354, 1967.
8. Barnard, R.J. & Weber, J.S. Carbon monoxide: a hazard to firefighters. Arch. Environ. Health 34:255-257, 1979.
9. Ayres, S.M., Evans, R., Licht, D., et al. Health effects of exposure to high concentrations of automotive emissions: study in bridge and tunnel workers in New York City. Arch. Environ. Health 27:168-178, 1973.
10. Cohen, S.I., Dorion, G., Goldsmith, J.R., et al. Carbon monoxide uptake by inspectors at a United States-Mexico border station. Arch. Environ. Health 22:47-54, 1971.
11. Aronow, W.S. & Isbell, M.W. Carbon monoxide effect on exercise-induced angina factors. Ann. Intern. Med. 79:392-5, 1973.
12. U.S. Environmental Protection Agency. Bureau of Science and Technology. Data Report/Automotive Network 1977. New York City, EPA, 1977, pp. 30-31.

13. Godin, G., Wright, G. & Shephard, R.J. Urban exposure to carbon monoxide. Arch. Environ. Health 25:305-313, 1972.
14. Silverman, F., Folinsbee, L.J., Barnard, J. & Shephard, R.J. Pulmonary function changes in ozone. Interaction of concentration and ventilation. J. Appl. Physiol. 41:859, 1976.
15. Folinsbee, L.J., Silverman, F., Shephard, R.J. Exercise responses following ozone exposure. J. Appl. Physiol. 38:996, 1975.
16. Folinsbee, L.J., Silverman, F., Shephard, R.J. Decrease of maximum work performance following ozone exposure. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 42:531, 1977.
17. Bedi, J.F., Folinsbee, L.J., Horvath, S.M. Pulmonary function changes of elite cyclists following a 1-hour exposure to 0.20 ppm ozone during heavy exercise. Med. Sci. Sports Exercise 15:112, 1973 (Abstract).
18. Adams, W.C., Scheledgle, E.S. Ozone and high ventilation effects on pulmonary function and endurance performance. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 55:805, 1983.
19. Silverman, F., Corey, P., Mintz, S., Olver, P., Hosein, R. A study of effects of ambient urban air pollution using personal samplers; a preliminary report. Environment International 8:311, 1983.
20. Shephard, R.J. Topographic and meteorological factors influencing air pollution in Cincinnati. A.M.A. Arch. Ind. Hlth 19:44, 1959.
21. Wright, G.R., Jewczyk, S., Onrot, J., et al. Carbon monoxide in the urban atmosphere. Arch. Environ. Health 30:123, 1975.
22. Silverman, F., Shephard, R.J., Folinsbee, L.J. Effect of physical activity on lung responses to acute ozone exposure. Proc. 4th International Clean Air Congress, Tokyo, Japan, May 15-20, 1977, p. 15-19.
23. Sheppard, D., Saisho, A., Nadel, J.A., Bonshey, H.A. Exercise increases sulfur dioxide-induced bronchoconstriction in asthmatic subjects. Am. Rev. Resp. Dis. 123:486, 1981.
24. Niinimaa, V., Cole, P., Mintz, S., Shephard, R.J. The

switching point from nasal to oronasal breathing. *Respir. Physiol.* 42:61, 1980.

25. Mintz, S., Hosein, H.R., Batten, B., Silverman, F. A personal sampler for three respiratory irritants. *JAPCA* 32:1068, 1982.
26. Silverman, F., Mintz, S., Olver, P., Corey, P., Hosein, H.R. et al. Human exposure to SO<sub>2</sub>, NO<sub>2</sub> and suspended particulate matter in Toronto, Canada. Published under the joint sponsorship of the United Nations Environment Programme and the World Health Organization, Geneva, EFP/82.38, 1982.
27. Silverman, F., Mintz, S., Hosein, R., Corey, P. To investigate and report on the health effects of ambient air pollution on asthmatics. Report submitted to Health and Welfare Canada, Environmental Health Directorate, December 1982.
28. Silverman, F., Pengelly, L.D., Mintz, S., Kerigan, A.T., Hosein, H.R., Corey, P., Goldsmith, C.H. Exposure estimates in assessing health effects of air pollution. *Environ. Monitoring & Assessment* 2:233, 1982.
29. Fletcher, R.A. A review of personal/portable monitors and samplers for airborne particles. *JAPCA* 34:1014, 1984.
30. West, P.W. & Gaeke, G.C. Fixation of sulphur dioxide as sulfitomercurate III and subsequent colorimetric determination. *Anal. Chem.* 28:1816, 1956.
31. National primary and secondary air quality standards. *Federal Register* 36:8186, 1971.
32. Mulik, J.D., Fuerst, R., Guyer, M., et al. Development of optimization of 24-hour manual methods for the collection and colorimetric analyses of atmospheric NO<sub>2</sub>. *Int. J. Environ. Anal. Chem.* 3:333, 1974.
33. Henderson, M., & Apthorp, G.A. Rapid method for estimation of carbon monoxide in blood. *Brit. Med. J.* 2:1853, 1960.
34. Shephard, R.J., Urch, B., Silverman, F., Corey, P. Interaction of ozone and cigarette smoke exposure. *Environ. Res.* 31:125, 1983.
35. Urch, R.B., Silverman, F., Corey, P., Shephard, R.J., Cole, P., and Goldsmith, L.J. Does suggestibility modify acute reactions to passive cigarette smoke

- exposure? Environ. Res. 47:34-47, 1988.
36. American Thoracic Society Statement-Snowbird Workshop on Standardization of Spirometry. Amer. Rev. Resp. Dis. 119:831, 1979.
  37. American Thoracic Society Questionnaire. Amer. Rev. Resp. Dis. 118:(part 2), 1978.
  38. Draper, H. and Smith, H. Applied regression analysis. Second edition, John Wiley and Sons, Toronto, 1988.
  39. Goldman, H.I. and Becklake, M.R. Respiratory function tests. Normal values at median altitudes and the prediction of normal results. Am. Rev. Respir. Dis. 79:457-467, 1959.
  40. Morris, J.F., Temple, W.P. and Koski, A. Normal values for the ratio of one-second forced expiratory volume to forced vital capacity. Am. Rev. Resp. Dis. 108:1000-1003, 1973.
  41. Lapp, N.L. and Hyatt, R.E. Some factors affecting the relationship of maximal expiratory flow to lung volume in health and disease. Dis. Chest 51:475-481, 1967.
  42. Cole, P. Nasal airflow resistance, Chapter 11, pp. 391-414, and Modification of inspired air, Chapter 12, pp. 415-445. In: "Respiratory Function of the Upper Airway". Ed: O.P. Mathew & G. Sant'Ambrogio. Marcel Dekker, Inc., New York, 1988.
  43. Dockery, D.W., Speizer, F.E., Stram, D.O., Ware, J.H., Spengler J.D. and Ferris, B.G. Effects of Inhalable Particles on Respiratory Health of Children. Am. Rev. Respir. Dis. 139(3): 584-594, 1989.
  44. Bates, D.V. and Sizto, R. Relationship between air pollutant levels and hospital admissions in southern Ontario. Can. J. Public Health 74:117-122, 1983.
  45. Bates, D.V. and Sizto, R. Air pollution and hospital admissions in southern Ontario: the acid summer haze effect. Environmental Research 43:317-331, 1987.
  46. Bates, D.V. and Sizto, R. The Ontario air pollution study: Identification of the causative agent. Environmental Health Perspectives 79:69-72, 1989.

TABLE 1A  
NUMBER OF REPEAT HEALTH TESTS ON RUNNERS

No. of Repeat Tests	No. of Runners	Cummulative No. of Runners	No. of Test-Sets *
14 and over	33	33	509
13	2	35	26
12	3	38	36
11	1	39	11
10	2	41	20
9	3	44	27
8	2	46	16
6	3	49	18
5	4	53	20
4	5	58	20
3	1	59	3
2	12	71	24
1	<u>10</u>	81	<u>10</u>
	81		740

\* Number of runners X number of repeat tests.



TABLE 1B

YEARLY SUMMARY OF HEALTH TESTING  
FOR RUNNERS WITH TWO OR MORE TESTS  
PLUS COMPLETE POLLUTION DATA

<u>Year</u>	<u>No. of Test-Sets</u>	<u>No. of Weeks of Testing</u>
1986	129	18
1987	313	29
1988	<u>270</u>	<u>28</u>
	712	75

TABLE 1C

SUMMARY OF RUN DISTANCE  
FOR RUNNERS WITH TWO OR MORE HEALTH TESTS  
PLUS COMPLETE POLLUTION DATA

<u>Distance (miles)*</u>	<u>No. of Runners</u>	<u>No. of Test-Sets</u>
2	1	1
4	2	2
5	2	2
6	52	204
7	3	3
8	46	149
9	1	2
10	58	337
11	2	2
12	5	7
14	3	3
	---	---
	175	712

\* Distance run, reported by runner.

Note: Runners may not have run the same distance each week.  
There was a total of 70 runners (54 male, 16 female)  
who had two or more health tests plus complete  
pollution data.

TABLE 2A

MEAN POLLUTANT CONCENTRATIONS FOR  
WEDNESDAY RUNS OVER 1987

AVERAGES FOR GAGE - 6 AND 10 MILE MOBILE SAMPLERS,  
GAGE - MOE STATIONARY SAMPLERS,  
AND MOE STATIONARY MONITORING SITES

		1987 (N=14 weeks)				
<u>POLLUTANT</u>		MEAN $\pm$		SD*	RANGE**	
NO <sub>2</sub>	(Gage - 6 mile)	34	$\pm$	9	16	- 52
NO <sub>2</sub>	(Gage - 10 mile)	30	$\pm$	10	9	- 52
NO <sub>2</sub>	(GAGE - Breadalbane)	30	$\pm$	10	15	- 50
NO <sub>2</sub>	(MOE - Breadalbane)	27	$\pm$	14	0	- 55
NO <sub>2</sub>	(MOE - Evans)	39	$\pm$	11	15	- 55
SO <sub>2</sub>	(Gage - 6 mile)	5	$\pm$	7	0	- 29
SO <sub>2</sub>	(Gage -10 mile)	6	$\pm$	7	0	- 25
SO <sub>2</sub>	(GAGE - Breadalbane)	4	$\pm$	6	0	- 21
SO <sub>2</sub>	(MOE - Breadalbane)	5	$\pm$	10	0	- 30
SO <sub>2</sub>	(MOE - Evans)	2	$\pm$	4	0	- 10
CO	(Gage - 6 mile)	4.3	$\pm$	1.5	2.2	- 7.4
CO	(Gage - 10 mile)	2.7	$\pm$	1.6	1.0	- 7.2
CO	(MOE - Breadalbane)	1.1	$\pm$	1.1	0.0	- 4.0
CO	(MOE - Evans)	1.4	$\pm$	0.5	1.0	- 2.0
RSP	(Gage - 6 mile)	104	$\pm$	37	41	- 159
RSP	(Gage - 10 mile)	72	$\pm$	16	44	- 93
RSP	(GAGE - Breadalbane)	55	$\pm$	22	28	- 110
SP	(MOE - Breadalbane)	69	$\pm$	25	27	- 101

\* Mean  $\pm$  standard deviation.

\*\* Range: lowest to highest level.

NO<sub>2</sub> reported as parts per billion (ppb). The Ontario MOE Ambient Air Quality Criterion (AAQC) for NO<sub>2</sub>, based on a 24-hr average is 200 micrograms/m<sup>3</sup> or 106 ppb (@ 25°C & 760 mmHg), and 400 micrograms/m<sup>3</sup> (213 ppb) based on a 1-hr average, the limiting effect based on health. The Ontario MOE 0.5-hr average Point of Impingement (POI) standard for NO<sub>2</sub> is 500 micrograms/m<sup>3</sup> or 267 ppb.

TABLE 2A  
(continued)

SO<sub>2</sub> reported as ppb. The Ontario MOE AAQC for SO<sub>2</sub>, based on a 24-hr average is 275 micrograms/m<sup>3</sup> (105 ppb), and 690 micrograms/m<sup>3</sup> (264 ppb) based on a 1-hr average, the limiting effect based on health and vegetation. The Ontario MOE 0.5-hr average POI standard for SO<sub>2</sub> is 830 micrograms/m<sup>3</sup> (317 ppb). CO reported as parts per million (ppm). The Ontario MOE AAQC for CO, based on a 8-hr average is 15700 micrograms/m<sup>3</sup> (13.7 ppm), the limiting effect based on high background levels from automobiles, and 36200 micrograms/m<sup>3</sup> (31.6 ppm) based on a 1-hr average, the limiting effect based on health. The Ontario MOE 0.5-hr average POI standard for CO is 6000 micrograms/m<sup>3</sup> (5.2 ppm).

SP reported as micrograms/m<sup>3</sup> (also RSP). The Ontario MOE AAQC for suspended particulate matter (d < 44 microns), based on a 24-hr average is 120 micrograms/m<sup>3</sup>, the limiting effect based on visibility. The Ontario MOE 0.5-hr average POI standard for SP is 100 micrograms/m<sup>3</sup>.

Note: The AAQC and POI Standards as of May 19, 1989 according to Ontario Regulation (O.R. 308)

- Gage: Gage mobile samplers (NO<sub>2</sub> & SO<sub>2</sub> - pump & impingers, RSP - pump & filter with cyclone).  
 6 mile - time-weighted average (1730 - 1830 hrs).  
 10 mile - time-weighted average (1730 - 1900 hrs).
- NO<sub>2</sub> - (TGS-ANSA method). SO<sub>2</sub> - (West & Gaeke method).  
 RSP - Respirable Suspended Particulate Matter).
- CO - Gage mobile electrochemical CO monitor (Industrial Scientific model CO260, with pump model SP200).  
 6 mile (average of 12 sites out & back along the route).  
 10 mile (average of 18 sites out & back along the route).
- Gage - Breadalbane: Gage stationary sampler at MOE site (Breadalbane). NO<sub>2</sub>, SO<sub>2</sub> & RSP sample method as above, time-weighted average (1700 - 1900 hrs).
- MOE - Breadalbane: Ontario Ministry of The Environment (MOE) pollutant monitoring network (Breadalbane site - downtown Toronto).
- MOE - Evans: MOE (Evans/Arnold Ave site - Etobicoke).  
 NO<sub>2</sub>, SO<sub>2</sub> & CO: Average of 1800 & 1900 hrs mean readings as below.  
 1800 hrs - mean of 12 5-minute averages 1700 - 1800 hrs.  
 1900 hrs - mean of 12 5-minute averages 1800 - 1900 hrs.  
 As of June, 1988, readings were taken every 2 seconds.
- SP: Suspended particulate matter (24-hour average, HiVol filter sample, 2400 hrs to 2400 hrs, reported as micrograms/m<sup>3</sup>).

TABLE 2B

MEAN POLLUTANT CONCENTRATIONS FOR  
WEDNESDAY RUNS OVER 1988

AVERAGES FOR GAGE - 6 AND 10 MILE MOBILE SAMPLERS,  
GAGE - MOE STATIONARY SAMPLERS,  
AND MOE STATIONARY MONITORING SITES

1988  
(N=20 weeks)

<u>POLLUTANT</u>		MEAN $\pm$ SD*		RANGE**	
NO <sub>2</sub>	(Gage - 6 mile)	37	$\pm$ 11	21	- 60
NO <sub>2</sub>	(Gage - 10 mile)	32	$\pm$ 10	14	- 47
NO <sub>2</sub>	(GAGE - Breadalbane)	29	$\pm$ 9	14	- 49
NO <sub>2</sub>	(MOE - Breadalbane)	29	$\pm$ 14	0	- 50
NO <sub>2</sub>	(MOE - Evans)	41	$\pm$ 20	10	- 100
SO <sub>2</sub>	(Gage - 6 mile)	3	$\pm$ 5	0	- 17
SO <sub>2</sub>	(Gage - 10 mile)	3	$\pm$ 4	0	- 14
SO <sub>2</sub>	(GAGE - Breadalbane)	5	$\pm$ 8	0	- 28
SO <sub>2</sub>	(MOE - Breadalbane)	8	$\pm$ 9	0	- 35
SO <sub>2</sub>	(MOE - Evans)	8	$\pm$ 13	0	- 60
CO	(Gage - 6 mile)	2.6	$\pm$ 1.3	0.8	- 5.0
CO	(Gage - 10 mile)	1.7	$\pm$ 1.1	0.4	- 3.6
CO	(MOE - Breadalbane)	0.7	$\pm$ 0.5	0.0	- 1.0
CO	(MOE - Evans)	1.1	$\pm$ 1.1	0.0	- 5.0
RSP	(Gage - 6 mile)	69	$\pm$ 37	8	- 142
RSP	(Gage - 10 mile)	55	$\pm$ 16	22	- 91
RSP	(GAGE - Breadalbane)	33	$\pm$ 13	7	- 56
SP	(MOE - Breadalbane)	68	$\pm$ 23	31	- 112

Same abbreviations as Table 2A

TABLE 2C

MEAN POLLUTANT CONCENTRATIONS FOR  
WEDNESDAY RUNS OVER 1987 and 1988

AVERAGES FOR GAGE - 6 AND 10 MILE MOBILE SAMPLERS,  
GAGE - MOE STATIONARY SAMPLERS,  
AND MOE STATIONARY MONITORING SITES

1987 and 1988  
(N=34 weeks)

<u>POLLUTANT</u>		MEAN $\pm$ SD*		RANGE**	
NO <sub>2</sub>	(Gage - 6 mile)	36	$\pm$ 10	16	- 60
NO <sub>2</sub>	(Gage - 10 mile)	31	$\pm$ 10	9	- 52
NO <sub>2</sub>	(GAGE - Breadalbane)	29	$\pm$ 9	14	- 50
NO <sub>2</sub>	(MOE - Breadalbane)	28	$\pm$ 14	0	- 55
NO <sub>2</sub>	(MOE - Evans)	40	$\pm$ 17	10	- 100
SO <sub>2</sub>	(Gage - 6 mile)	4	$\pm$ 6	0	- 29
SO <sub>2</sub>	(Gage - 10 mile)	4	$\pm$ 5	0	- 25
SO <sub>2</sub>	(GAGE - Breadalbane)	5	$\pm$ 8	0	- 28
SO <sub>2</sub>	(MOE - Breadalbane)	6	$\pm$ 10	0	- 35
SO <sub>2</sub>	(MOE - Evans)	5	$\pm$ 11	0	- 60
CO	(Gage - 6 mile)	3.3	$\pm$ 1.6	0.8	- 7.4
CO	(Gage - 10 mile)	2.1	$\pm$ 1.4	0.4	- 7.2
CO	(MOE - Breadalbane)	0.9	$\pm$ 0.8	0.0	- 4.0
CO	(MOE - Evans)	1.2	$\pm$ 0.9	0.0	- 5.0
RSP	(Gage - 6 mile)	84	$\pm$ 40	8	- 159
RSP	(Gage - 10 mile)	62	$\pm$ 18	22	- 93
RSP	(GAGE - Breadalbane)	42	$\pm$ 20	7	- 110
SP	(MOE - Breadalbane)	69	$\pm$ 23	27	- 112

Same abbreviations as Table 2A

TABLE 3A

MEAN NO<sub>2</sub> CONCENTRATIONS FOR  
WEDNESDAY RUNS OVER 1986 -1988

AVERAGES FOR GAGE - 6 AND 10 MILE MOBILE SAMPLERS,  
GAGE - MOE STATIONARY SAMPLERS,  
AND MOE STATIONARY MONITORING SITES

SITE	WEEKS	NO <sub>2</sub> (ppb)		
		MEAN	± SD	RANGE
(a) Gage (6 mile)	55	36	± 11	16 - 69
(b) Gage (10 mile)	70	33	± 14	6 - 78
(c) Gage - Breadalbane	65	29	± 14	2 - 87
(d) B-MOE (1800 hrs)	73	29	± 13	0 - 70
(e) B-MOE (1900 hrs)	72	30	± 15	0 - 70
(f) B-MOE (mean of d + e)	73	29	± 14	0 - 70
(g) E-MOE (1800 hrs)	74	37	± 27	10 - 210
(h) E-MOE (1900 hrs)	73	40	± 26	0 - 210
(i) E-MOE (mean of g + h)	74	38	± 26	5 - 210

Same abbreviations as Table 2A.

B-MOE: Breadalbane MOE site.

E-MOE: Evans Ave. MOE site.

1800 hrs - mean of 12 5-minute averages 1700 - 1800 hrs.

1900 hrs - mean of 12 5-minute averages 1800 - 1900 hrs.

As of June, 1988 readings were taken every 2 seconds.

TABLE 3B

MEAN SO<sub>2</sub> CONCENTRATIONS FOR  
WEDNESDAY RUNS OVER 1986 -1988

AVERAGES FOR GAGE - 6 AND 10 MILE MOBILE SAMPLERS,  
GAGE - MOE STATIONARY SAMPLERS,  
AND MOE STATIONARY MONITORING SITES

SITE	WEEKS	SO <sub>2</sub> (ppb)		
		MEAN	± SD	RANGE
(a) Gage (6 mile)	55	6	± 8	0 - 37
(b) Gage (10 mile)	71	7	± 8	0 - 44
(c) Gage - Breadalbane	64	4	± 7	0 - 28
(d) B-MOE (1800 hrs)	74	4	± 10	0 - 50
(e) B-MOE (1900 hrs)	72	4	± 8	0 - 40
(f) B-MOE (mean of d + e)	74	4	± 8	0 - 35
(g) E-MOE (1800 hrs)	71	5	± 16	0 - 120
(h) E-MOE (1900 hrs)	71	6	± 16	0 - 120
(i) E-MOE (mean of g + h)	72	6	± 16	0 - 120

Same abbreviations as Table 2A, 3A.

TABLE 3C

MEAN CO CONCENTRATIONS FOR  
WEDNESDAY RUNS OVER 1986 -1988

AVERAGES FOR GAGE - 6 AND 10 MILE MOBILE SAMPLERS,  
AND MOE STATIONARY MONITORING SITES

SITE	WEEKS	CO (ppm)		
		MEAN $\pm$	SD	RANGE
(a) Gage (6 mile)*	72	3.8 $\pm$	1.9	0.6 - 9.1
(b) Gage (10 mile)	73	2.7 $\pm$	1.6	0.4 - 8.1
(c) B-MOE (1800 hrs)	72	0.9 $\pm$	0.7	0.0 - 4.0
(d) B-MOE (1900 hrs)	72	1.0 $\pm$	0.7	0.0 - 4.0
(e) B-MOE (mean of c + d)	73	1.0 $\pm$	0.7	0.0 - 4.0
(f) E-MOE (1800 hrs)	72	1.4 $\pm$	1.5	0.0 - 11.0
(g) E-MOE (1900 hrs)	71	1.4 $\pm$	1.5	0.0 - 11.0
(h) E-MOE (mean of f + g)	72	1.4 $\pm$	1.5	0.0 - 11.0

Gage: Gage mobile electrochemical CO monitor (Industrial Scientific model CO260, with pump model SP200).  
6 mile (average of 12 sites out & back along the route).  
10 mile (average of 18 sites out & back along the route).

\* In 1986 there was only one CO monitor (10 mile). Six mile mean values were calculated using recorded 10 mile "out" values (University Settlement House out to the 3 - mile turn point), & estimated values for 3 - mile "back" (3 - mile turn point back to University Settlement House) values (using the ratio of "out/back" recorded in 1987 when there was a 6 and 10 mile CO monitor). Since the ratio of "out/back" was fairly constant over 1987, the assumption was made that this was the case in 1986, as the running route was the same.

Same abbreviations as Tables 2A, 3A.



TABLE 3D  
MEAN CO CONCENTRATIONS FOR SITES ALONG  
6 MILE WEDNESDAY RUN ROUTE OVER 1987 - 1988

Site	Out (weeks)	Back (weeks)
University Settlement House	1.2 $\pm$ 1.6 (53)	1.1 $\pm$ 1.0 (54)
Queen and Soho	4.1 $\pm$ 3.8 (52)	3.6 $\pm$ 3.1 (52)
King and Peter	5.1 $\pm$ 4.3 (54)	2.8 $\pm$ 3.1 (50)
Front and Peter	6.1 $\pm$ 7.9 (37)	3.4 $\pm$ 5.8 (10)
Front and Spadina	8.3 $\pm$ 7.2 (52)	4.8 $\pm$ 3.1 (52)
Spadina Bridge	3.2 $\pm$ 1.9 (25)	2.6 $\pm$ 2.6 (28)
QEW and Spadina	9.7 $\pm$ 6.2 (26)	8.4 $\pm$ 4.7 (30)
Queen's Quay and Spadina	4.0 $\pm$ 2.4 (24)	3.6 $\pm$ 2.6 (8)
Lakeshore and Bathurst	1.6 $\pm$ 1.5 (24)	2.3 $\pm$ 1.6 (21)
Tip Top Taylors (Lakeshore)	2.8 $\pm$ 2.3 (54)	1.4 $\pm$ 1.3 (53)
War Plane at Ontario Place	1.1 $\pm$ 1.1 (54)	1.0 $\pm$ 1.0 (53)
Remembrance Drive (3 mile turn)	2.1 $\pm$ 1.8 (54)	2.2 $\pm$ 2.2 (30)
Mean CO concentration (ppm) (all sites averaged)	3.8 $\pm$ 2.0 (54)	2.9 $\pm$ 1.4 (54)
Peak CO concentration (ppm)	12.1 $\pm$ 8.2 (54)	8.3 $\pm$ 4.4 (54)
Mean CO concentration - all sites out and back:	3.4 $\pm$ 1.6 ppm (54)	

TABLE 3E  
MEAN CO CONCENTRATIONS FOR SITES ALONG  
10 MILE WEDNESDAY RUN ROUTE OVER 1986 - 1988

Site	Out (weeks)	Back (weeks)
University Settlement House	1.4 $\pm$ 1.9 (71)	1.0 $\pm$ 1.4 (65)
Queen and Soho	5.3 $\pm$ 8.3 (69)	2.8 $\pm$ 2.5 (68)
King and Peter	4.4 $\pm$ 4.3 (71)	1.7 $\pm$ 1.6 (66)
Wellington and Peter	3.2 $\pm$ 4.1 (16)	1.5 $\pm$ 2.1 (20)
Front and Peter	6.0 $\pm$ 6.3 (54)	2.7 $\pm$ 2.4 (32)
Front and Spadina	8.3 $\pm$ 7.5 (68)	4.5 $\pm$ 3.4 (65)
Front and Portland	1.0 $\pm$ 1.1 (25)	1.1 $\pm$ 1.7 (21)
Spadina Bridge underpass	4.0 $\pm$ 2.8 (44)	2.8 $\pm$ 2.5 (45)
QEW and Spadina	8.4 $\pm$ 5.2 (45)	5.1 $\pm$ 3.8 (41)
Bathurst Bridge overpass	3.0 $\pm$ 2.9 (27)	1.2 $\pm$ 1.1 (22)
Queen's Quay and Spadina	4.7 $\pm$ 3.6 (41)	4.4 $\pm$ 6.2 (26)
Canada Malting	1.6 $\pm$ 1.6 (24)	3.3 $\pm$ 2.8 (24)
Lakeshore and Bathurst	3.6 $\pm$ 2.9 (27)	3.4 $\pm$ 4.3 (25)
Tip Top Taylors (Lakeshore)	2.2 $\pm$ 2.3 (53)	1.6 $\pm$ 4.7 (53)
War Plane at Ontario Place	0.8 $\pm$ 1.4 (55)	1.0 $\pm$ 1.2 (54)
Remembrance Dr. (3 mile turn)	1.8 $\pm$ 2.0 (73)	1.6 $\pm$ 1.6 (70)
Lakeshore Blvd. (bleachers)	1.6 $\pm$ 2.1 (70)	1.4 $\pm$ 1.7 (68)
Aquatic Drive	1.1 $\pm$ 1.4 (71)	0.9 $\pm$ 1.2 (68)
Tennis courts (4 mile turn)	1.3 $\pm$ 1.4 (54)	1.1 $\pm$ 1.5 (54)
Boulevard Club	1.5 $\pm$ 1.6 (71)	1.3 $\pm$ 1.6 (66)
Budapest Park	1.0 $\pm$ 1.5 (54)	1.0 $\pm$ 1.4 (53)
Sunnyside Pool (5 mile turn)	1.5 $\pm$ 2.2 (70)	0.8 $\pm$ 1.0 (11)
Mean CO concentration (ppm) (all sites averaged)	3.2 $\pm$ 2.0 (73)	2.1 $\pm$ 1.5 (73)
Peak CO concentration (ppm)	12.7 $\pm$ 9.4 (73)	7.6 $\pm$ 5.6 (73)
Mean CO concentration - all sites out and back:	2.7 $\pm$ 1.6 ppm (73)	

TABLE 4A

MEAN TEMPERATURE FOR  
WEDNESDAY RUNS OVER 1986 -1988

AVERAGES FOR GAGE 10 MILE MOBILE MONITOR,  
MOE AND AES STATIONARY MONITORING SITES

SITE	WEEKS	Temperature (Degrees Celcius)		
		MEAN $\pm$	SD	RANGE
(a) Gage (10 mile)	73	18 $\pm$ 7		5 - 31
(b) E-MOE (1800 hrs)	74	17 $\pm$ 7		2 - 33
(c) E-MOE (1900 hrs)	73	17 $\pm$ 7		2 - 32
(d) E-MOE (mean of b + c)	74	17 $\pm$ 7		2 - 33
(e) TC-AES (mean 1800,1900 hrs)	75	18 $\pm$ 7		2 - 32

Same abbreviations as Table 3A.

Gage: Gage mobile temperature/humidity monitor (Jenway model 5500, wet bulb/dry bulb). Mean of 4 sites along the running route.

TC-AES: Environment Canada - Atmospheric Environment Services  
Trinity College site, Queen's Park.

Note: No Gage mobile data was collected May 6 & Nov. 11, 1987,  
TC-AES data was used May 6:Temp=23°C, Nov. 11:Temp=2°C.

TABLE 4B

MEAN RELATIVE HUMIDITY FOR  
WEDNESDAY RUNS OVER 1987 -1988

AVERAGE FOR GAGE 10 MILE MOBILE MONITOR  
AND AES STATIONARY MONITORING SITE

SITE	WEEKS	Relative Humidity (Percent)		
		MEAN $\pm$	SD	RANGE
Gage (10 mile)	54	60 $\pm$ 14		34 - 88
TC-AES	54	58 $\pm$ 16		26 - 95

Same abbreviations as Table 4A.

Gage: Gage mobile temperature/humidity monitor (Jenway model 5500, wet bulb/dry bulb).

Mean of 4 sites along the running route.

Note: No Gage mobile data was collected in 1986,  
TC-AES data was used: N= 18, mean RH= 63  $\pm$  17%, range= 32 - 88%.  
No Gage mobile data was collected May 6, July 15 & Nov. 11, 1987.  
TC-AES data was used May 6:RH=25%, July 15:RH=51%, Nov. 11:RH=44%.

TABLE 5A

AVERAGE SYMPTOM AND POLLUTION RATINGS  
WEDNESDAY RUNS OVER 1986 - 1988

N	SYMPTOM	VARIABLE	MEAN RATING $\pm$ SD		RANGE	
700	69%*	Pollution Rating	3.3	$\pm$ 1.6	0.0	- 10.0
581	67%*	Leg Fatigue	3.3	$\pm$ 1.8	0.0	- 10.0
581	61%*	Breathing Fatigue	2.9	$\pm$ 1.6	0.0	- 10.0
464	72%**	Nasal Discharge	1.9	$\pm$ 1.8	0.0	- 10.0
464	70%**	Sputum/Phlegm	1.8	$\pm$ 1.8	0.0	- 10.0
464	68%**	Nasal Stuffiness	1.7	$\pm$ 1.7	0.0	- 8.0
464	54%**	Sore/Dry Throat	1.2	$\pm$ 1.5	0.0	- 7.0
464	60%**	Eye Irritation	1.1	$\pm$ 1.4	0.0	- 8.0
464	38%**	Cough	0.8	$\pm$ 1.4	0.0	- 7.0

N: Number of test-sets (see Table 1A).

MEAN RATING: After the run, subjects were asked to subjectively rate their symptoms from the above list, and to rate the pollution during the run using a modified Borg rating scale as detailed below:

0	- Nothing at all.	5	- Strong.
0.5	- Very, very weak- (just noticeable).	6	
1	- Very weak.	7	- Very strong.
2	- Weak.	8	
3	- Moderate.	9	
4	- Somewhat strong.	10	- Very, very strong (almost max). Maximal.

SYMPTOM: \* Percentage reporting Moderate (3) rating or higher.

\*\* Percentage reporting any trace (0.5) rating or higher.

TABLE 5B

AVERAGE SYMPTOM AND POLLUTION RATINGS  
WEDNESDAY RUNS OVER 1986 - 1988  
FOR 6, 8 AND 10 MILE GROUPS

VARIABLE	MEAN RATINGS $\pm$ SD		
	6 mile	8 mile	10 mile
(a) Pollution Rating	3.4 $\pm$ 1.6	3.2 $\pm$ 1.5	3.2 $\pm$ 1.6
(b) Leg Fatigue	3.1 $\pm$ 1.8	3.2 $\pm$ 1.8	3.4 $\pm$ 1.8
(c) Breathing Fatigue	2.9 $\pm$ 1.6	2.7 $\pm$ 1.5	3.1 $\pm$ 1.7
(d) Nasal Discharge	1.8 $\pm$ 1.8	1.9 $\pm$ 1.8	2.0 $\pm$ 1.8
(e) Sputum/Phlegm	2.0 $\pm$ 1.9	1.8 $\pm$ 1.8	1.8 $\pm$ 1.8
(f) Nasal Stuffiness	1.6 $\pm$ 1.8	1.7 $\pm$ 1.6	1.7 $\pm$ 1.7
(g) Sore/Dry Throat	1.4 $\pm$ 1.6	1.2 $\pm$ 1.6	1.1 $\pm$ 1.4
(h) Eye Irritation	1.1 $\pm$ 1.3	1.1 $\pm$ 1.2	1.2 $\pm$ 1.6
(i) Cough	0.9 $\pm$ 1.4	0.8 $\pm$ 1.4	0.8 $\pm$ 1.4

Same abbreviations as Table 5A.

6 mile: 2, 4, 5 or 6 miles (N= 209:a, 174:a & c, 134:d to i)

8 mile: 7, 8 or 9 miles (N= 153:a, 137:b & c, 110:d to i)

10 mile: 10, 12 or 14 miles (N= 338:a, 270:b & c, 220:d to i)

TABLE 6A  
BACKGROUND ANTHROPOMETRIC AND PULMONARY FUNCTION  
TEST RESULTS (THE GAGE RESEARCH INSTITUTE)

ANTHROPOMETRIC	MALES (N=34)	FEMALES (N=11)	ALL (N=45)
Age (yrs)	38 $\pm$ 9	34 $\pm$ 10	37 $\pm$ 9
Height (cm)	174.6 $\pm$ 6.3	163.8 $\pm$ 7.5	172.0 $\pm$ 8.0
Body Mass(kg)	69.3 $\pm$ 6.9	55.4 $\pm$ 9.6	65.9 $\pm$ 9.6

Note: Values are means  $\pm$  standard deviation.

PULMONARY FUNCTION	MALES (N=33)	FEMALES (N=11)	ALL (N=44)
FVC (ml)	5243 (113)	3897 (112)	4914 (113)
FEV <sub>1</sub> (ml)	4202 (106)	3225 (109)	3964 (107)
FEV <sub>1</sub> /FVC (%)	80	83	81
PEFR (ml/sec)	11044 (N=33)	7794	10231 (N=44)
FEF <sub>50</sub> (ml/sec)	4962 (108)	4029 (117)	4734 (110)
FEF <sub>75</sub> (ml/sec)	1758 (75)	1509 (86)	1697 (78)

Note: Values are means (percent of predicted normal values).  
FVC = forced vital capacity.  
FEV<sub>1</sub> = forced 1-second expiratory volume.  
PEFR = peak expiratory flow rate.  
FEF<sub>50</sub> = forced expiratory flow at 50% of FVC.  
FEF<sub>75</sub> = forced expiratory flow at 75% of FVC.

Percent predicted according to:  
FVC: Goldman and Becklake (1959).  
FEV<sub>1</sub>: Morris, Temple and Koski (1973).  
FEF<sub>50</sub>, FEF<sub>75</sub>: Lapp and Hyatt (1967).

LUNG VOLUMES	MALES (N=31)	FEMALES (N=9)	ALL (N=40)
TLC (ml)	7093 (106)	5260 (91)	6645 (92)
TGV (ml)	3451 (92)	2661 (91)	3258 (92)
RV (ml)	1850 (93)	1363 (88)	1731 (92)
R <sub>aw</sub> (cm H <sub>2</sub> O/L/sec)	1.24 (N=31)	1.43 (N=9)	1.28 (N=40)

Note: Values are means (percent of predicted normal values according to Goldman and Becklake (1959)).  
Lung volumes using a Collins pressure plethysmograph.  
TLC = total lung capacity.  
TGV = thoracic gas volume.  
RV = residual volume.  
R<sub>aw</sub> = airway resistance.

DIFFUSION	MALES (N=18)	FEMALES (N=2)	ALL (N=20)
D <sub>LCO</sub> (mlCO/min/mmHg)	36 (100)	25 (103)	35 (100)

Note: Values are means (percent of predicted normal values according to Gaensler and Smith (1973)).  
D<sub>LCO</sub> Single breath carbon monoxide diffusion capacity of the lung using a Collins modular system.

TABLE 6B

BASELINE PULMONARY FUNCTION AND COHb% TEST RESULTS  
PRE-WEDNESDAY RUNS OVER 1986 - 1988

VARIABLE	N	MEAN	±	SD	RANGE	
FVC (ml)	683	4991	±	896	2910	to 8980
FEV <sub>1</sub> (ml)	683	4031	±	690	2240	to 7120
IC (ml)	683	3376	±	720	1740	to 6720
PEFR (ml/sec)	683	9709	±	1677	5150	to 14850
FEF <sub>50</sub> (ml/sec)	683	4929	±	1290	1930	to 8890
FEF <sub>75</sub> (ml/sec)	683	1982	±	674	590	to 4610
COHb%	663	0.40	±	0.23	0.03	to 2.25

Note: Same abbreviations as Table 6A.

N: Number of test-sets (see Table 1A).

IC: inspiratory capacity.

COHb%: oxygen rebreathed estimate of blood carboxyhaemoglobin percent.

TABLE 6C

MEAN PULMONARY FUNCTION AND COHb% ABSOLUTE CHANGE  
WEDNESDAY RUNS OVER 1986 - 1988

VARIABLE	N	MEAN	±	SD*	RANGE	
FVC (ml)	683	- 27	±	166	- 770	to 600
FEV <sub>1</sub> (ml)	683	- 5	±	158	- 730	to 530
IC (ml)	683	- 171	±	286	- 2060	to 920
PEFR (ml/sec)	682	- 132	±	511	- 2380	to 3280
FEF <sub>50</sub> (ml/sec)	683	- 22	±	514	- 2340	to 1960
FEF <sub>75</sub> (ml/sec)	683	- 40	±	334	- 1840	to 1660
COHb%	655	- 0.07	±	0.13	- 1.54	to 0.26

\* Mean absolute change (post minus prerun)

A negative value indicates a decrease over the run.

Note: Same abbreviations as Table 6B.

TABLE 6D

MEAN PULMONARY FUNCTION AND COHb% CHANGES (%)  
WEDNESDAY RUNS OVER 1986 - 1988

VARIABLE	N	PERCENT**	±	SD	RANGE	
FVC	683	- 0.5	±	3.3	- 15.8	to 13.6
FEV <sub>1</sub>	683	- 0.2	±	3.9	- 17.9	to 15.4
IC	683	- 4.7	±	8.2	- 43.8	to 34.7
PEFR	682	- 1.3	±	5.3	- 22.7	to 36.5
FEF <sub>50</sub>	683	- 0.1	±	10.3	- 36.6	to 40.0
FEF <sub>75</sub>	683	- 3.6	±	17.7	- 60.0	to 101.2
COHb%	655	- 10.3	±	40.9	- 87.1	to 527.8

\*\* Mean percent change (post run minus prerun/prerun) x 100

Note: Same abbreviations as Table 6B.

TABLE 6E  
 MEAN PULMONARY FUNCTION AND COHb% CHANGES (%)  
 WEDNESDAY RUNS OVER 1986 - 1988  
 FOR 6, 8 AND 10 MILE GROUPS

VARIABLE	PERCENT CHANGE**		
	6 mile	8 mile	10 mile
(a) FVC	0.1 ± 3.2	- 0.5 ± 3.0	- 0.8 ± 3.5
(b) FEV <sub>1</sub>	0.2 ± 4.2	- 0.2 ± 3.5	- 0.4 ± 4.0
(c) IC	- 5.3 ± 7.7	- 5.0 ± 9.0	- 4.3 ± 8.1
(d) PEFR	- 1.8 ± 5.2	- 1.0 ± 5.5	- 1.1 ± 5.2
(e) FEF <sub>50</sub>	- 0.7 ± 9.8	- 0.7 ± 10.2	- 1.0 ± 10.6
(f) FEF <sub>75</sub>	0.9 ± 18.0	1.9 ± 16.0	5.9 ± 17.9
(g) COHb%	- 9.1 ± 40.3	- 12.7 ± 27.5	- 9.9 ± 46.1

\*\* Mean percent change (post run minus prerun/prerun) x 100

6 mile (N= 200:a to f, 188:g)

8 mile (N= 149:a to f, 146:g)

10 mile (N= 334:a,b,d,e & f, 333:c, 321:g)

Same abbreviations as Table 6D.

TABLE 7 FREQUENCY DISTRIBUTIONS OF THE SYMPTOM SEVERITY VARIABLES  
N = 464

<u>RATING*</u>	SORE/DRY THROAT (%)	COUGH (%)	SPUTUM/ PHLEGM (%)	STUFFY NOSE (%)	NASAL DISCHARGE (%)	EYE IRRITATION (%)
0.0	46.1	62.0	30.4	31.7	27.6	41.2
0.5	4.5	3.9	6.7	4.7	3.9	7.5
1.0	14.2	9.9	12.5	15.7	13.1	20.3
2.0	16.2	12.3	14.4	14.9	18.3	14.0
3.0	9.5	6.5	19.6	21.6	22.0	10.6
4.0	4.7	2.2	7.8	6.0	8.0	3.4
>5.0	<u>4.8</u> 100.0	<u>3.2</u> 100.0	<u>8.6</u> 100.0	<u>5.4</u> 100.0	<u>7.1</u> 100.0	<u>3.0</u> 100.0

\* The coding for the rating scale:

- 0 - Nothing at all
- 0.5 - Very, very weak (just noticeable)
- 1 - Very weak
- 2 - Weak
- 3 - Moderate
- 4 - Somewhat strong
- 5 - Strong
- 6
- 7 - Very strong
- 8
- 9
- 10 - Very, very strong (almost maximum)  
Maximal.

TABLE 8                      FREQUENCY DISTRIBUTION OF THE FATIGUE VARIABLES  
AND THE POLLUTION RATING VARIABLE  
N = 581

<u>RATING*</u>	BREATHING FATIGUE (%)	LEG FATIGUE (%)	POLLUTION RATING (%)
0.0	5.3	4.8	0.7
0.5	2.8	2.6	1.9
1.0	10.2	7.2	7.7
2.0	21.2	18.4	20.3
3.0	30.5	27.7	34.9
4.0	13.8	17.7	17.0
>5.0	<u>16.2</u> 100.0	<u>21.6</u> 100.0	<u>17.5</u> 100.0

\* The coding for the rating scale:

0	-	Nothing at all
0.5	-	Very, very weak (just noticeable)
1	-	Very weak
2	-	Weak
3	-	Moderate
4	-	Somewhat strong
5	-	Strong
6		
7	-	Very strong
8		
9		
10	-	Very, very strong (almost maximum) Maximal.



TABLE 9      MULTIPLE REGRESSION EQUATIONS FOR SYMPTOMS AND EXPOSURES  
OBTAINED FROM BACKWARD STEPWISE MULTIPLE LINEAR REGRESSION

SORE/DRY THROAT	=	0.18 x POLLUTE (0.001)*	-	0.014 x AVRH (0.002)	
COUGH	=	0.09 x MEANCO (0.04)	-	0.01 x AVRH (0.02)	- 0.033 x MEANRBCO (0.08)
SPUTUM/ PHLEGM	=	0.19 x POLLUTE (0.001)	-	0.045 x AVTEMP (0.0001)	- 0.010 x AVRH (0.04)
		+ 0.01 x SO <sub>2</sub> (0.06)			
STUFFY NOSE	=	0.27 x POLLUTE (0.0001)	-	0.049 x AVTEMP (0.0001)	+ 0.0091 x SO <sub>2</sub> (0.04)
		-0.047 x MEANRBCO (0.01)			
NASAL DISCHARGE	=	0.28 x POLLUTE (0.0001)	-	0.08 x AVTEMP (0.0001)	- 0.012 x AVRH (0.01)
BREATHING FATIGUE	=	0.26 x POLLUTE (0.0001)	-	0.08 x MEANCO (0.05)	
LEG FATIGUE	=	0.13 x POLLUTE (0.06)	-	0.10 x MEANCO (0.10)	+ 0.029 x AVTEMP (0.02)
EYE IRRITATION	=	0.19 x POLLUTE (0.0002)			

The predictor list includes Gage pollutant and environmental measures, COHb, and the pollution rating scale (POLLUTE).

\* Bracketed values are the p values associated with Student's t test of the hypothesis that the partial regression coefficient is zero.

For the symptom variables LEG and BREATHING FATIGUE N = 491, for all others N = 423.

Table 9 continued:

UNITS OF MEASURE FOR VARIABLES

For symptoms and POLLUTE (pollution rating) see rating scale in Table 8.

AVRH - average relative humidity (%)

AVTEMP - average temperature (°C)

MEANCO & PEAKCO (ppm)

MEANRBCO - mean rebreathe CO (average of prerun and post run) bag concentration (ppm)

DRBCO - rebreathe CO (post run - rebreathe CO prerun), bag concentration (ppm)

$$\text{COHb\%} = \frac{22638 \times \text{bagCO (ppm)}}{[.0231 \times \text{bagCO (ppm)}] + 79}$$

SO<sub>2</sub>, NO<sub>2</sub> & RSP (micrograms/m<sup>3</sup>)

Note: The Gage "personal" pollutant concentrations used in the regression equations were matched according to the distance the individual ran, to the 6 and 10 mile (bicycle) concentrations, as follows:

<u>Distance Run</u>	<u>Concentration Used</u>
Less than or equal to 6 miles	6 mile
Greater than or equal to 10 miles	10 mile
Less than 10 miles or greater than 6 miles	mean of 6 and 10 mile

TABLE 10      MULTIPLE REGRESSION EQUATIONS FOR SYMPTOMS AND EXPOSURES  
OBTAINED FROM BACKWARD STEPWISE MULTIPLE LINEAR REGRESSION

SORE/DRY THROAT	=	-0.014 x AVRH (0.002)			
COUGH	=	-0.010 x AVRH (0.02)	- 0.0074 x SO <sub>2</sub> (0.10)	+ 0.11 x MEANCO (0.01)	
		- 0.034 x MEANRBCO (0.08)			
SPUTUM/ PHLEGM	=	-0.0091 x AVRH (0.05)	+ 0.0096 x SO <sub>2</sub> (0.06)	- 0.037 x AVTEMP (0.0002)	
STUFFY NOSE	=	0.0062 x NO <sub>2</sub> (0.06)	+ 0.016 x PEAKCO (0.04)	- 0.04 x AVTEMP (0.0001)	
		- 0.05 x MEANRBCO (0.02)			
NASAL DISCHARGE	=	-0.07 x AVTEMP (0.0001)	- 0.011 x AVRH (0.02)		
BREATHING FATIGUE	=	0.019 x AVTEMP (0.04)			
LEG FATIGUE	=	0.042 x AVTEMP (0.0002)			
EYE IRRITATION	=	0.005 x NO <sub>2</sub> (0.12)	- 0.10 x MEANCO (0.08)	+ 0.024 x PEAKCO (0.02)	
POLLUTION RATING	=	0.009 x NO <sub>2</sub> (0.001)	+ 0.14 x MEANCO (0.002)	+ 0.049 x AVTEMP (0.0001)	

Only Gage pollutant and environmental measures and COHb are included in the predictor list.

Same abbreviations as Table 9.

TABLE 11      MULTIPLE REGRESSION EQUATIONS FOR PULMONARY FUNCTION  
VARIABLES AND EXPOSURES OBTAINED FROM BACKWARD STEPWISE  
MULTIPLE LINEAR REGRESSION

PEFPOST	=	-0.007 x AVTEMP	+	0.003 x AVRH	
		(0.06)		(0.06)	
DIC	=	0.008 x MEANRBCO	-	0.015 x POLLUTE	
		(0.02)		(0.12)	
ICPOST	=	0.0059 x MEANRBCO	-	0.022 x POLLUTE	
		(0.05)		(0.005)	
DFVC	=	0.0005 x NO <sub>2</sub>	-	0.002 x AVTEMP	
		(0.08)		(0.02)	
FVCPOST	=	0.0009 x NO <sub>2</sub>			
		(0.007)			
FEV <sub>1</sub> POST	=	0.0044 x AVTEMP	+	0.003 x MEANRBCO	- 0.0076 x POLLUTE
		(0.0001)		(0.10)	(0.11)
DFEF <sub>50</sub>	=	0.006 x AVTEMP			
		(0.04)			
FEF <sub>50</sub> POST	=	0.0053 x AVTEMP	-	0.012 x MEANRBCO	- 0.015 x DRBCO
		(0.08)		(0.08)	(0.01)
DFEF <sub>75</sub>	=	-0.001 x NO <sub>2</sub>	-	0.021 x MEANCO	
		(0.10)		(0.04)	
FEF <sub>75</sub> POST	=	-0.002 x NO <sub>2</sub>	+	0.002 x SO <sub>2</sub>	+ 0.005 x AVTEMP
		(0.001)		(0.04)	(0.02)

The predictor list includes Gage pollutant and environmental measures, COHb, and the pollution rating scale (POLLUTE).

Bracketed values are the p values associated with Student's t test of the hypothesis that the partial regression coefficient is zero.

For all analyses N = 539.

Table 11 continued:

UNITS OF MEASURE FOR VARIABLES:

See Tables 6A and 6B for definition of pulmonary function variables.

POST = post run value

D = mean absolute change (post run - prerun)

FVC, FEV<sub>1</sub>, IC (litres)

FEF<sub>50</sub>, FEF<sub>75</sub>, PEF (litres/sec)

Predictor variables have the same units as Table 9.

TABLE 12      MULTIPLE REGRESSION EQUATIONS FOR PULMONARY FUNCTION  
VARIABLES AND EXPOSURES OBTAINED FROM BACKWARD STEPWISE  
MULTIPLE LINEAR REGRESSION

PEFPOST	=	-0.007 x AVTEMP	+	0.003 x AVRH
		(0.05)		(0.06)
DIC	=	0.0083 x MEANRBCO		
		(0.02)		
ICPOST	=	-0.003 x AVTEMP		
		(0.04)		
DFVC	=	-0.0018 x AVTEMP		
		(0.03)		
FVCPOST	=	0.0009 x NO <sub>2</sub>		
		(0.01)		
FEV <sub>1</sub> POST	=	0.0041 x AVTEMP	+	0.0026 x MEANRBCO
		(0.0001)		(0.14)
DFEF <sub>50</sub>	=	0.006 x AVTEMP		
		(0.06)		
FEF <sub>50</sub> POST	=	0.006 x AVTEMP	-	0.01 x DRBCO
		(0.07)		(0.07)
FEF <sub>75</sub>	=	-0.001 x NO <sub>2</sub>	-	0.02 x MEANCO
		(0.14)		(0.05)
FEF <sub>75</sub> POST	=	0.004 x AVTEMP	-	0.002 x NO <sub>2</sub> + 0.001 x SO <sub>2</sub>
		(0.05)		(0.005) (0.12)

Only Gage pollutant measures are included in the predictor list.

Bracketed values are the p values associated with Student's t test of the hypothesis that the partial regression coefficient is zero.

For all analyses N = 547.

Same abbreviations as Table 11.

TABLE 13      MULTIPLE REGRESSION EQUATIONS FOR SYMPTOMS AND EXPOSURE:  
OBTAINED FROM BACKWARD STEPWISE MULTIPLE LINEAR REGRESSION

SORE/DRY THROAT	=	0.16 x POLLUTE (0.04)	-	0.01 x AVRH (0.02)	
COUGH	=	0.11 x POLLUTE (0.07)	-	0.01 x SO <sub>2</sub> (0.05)	+ 0.12 x MEANCO (0.04)
		-0.0089 x AVRH (0.06)	-	0.055 x MEANRBCO (0.03)	+ 9.6 x BNO (0.13)
SPUTUM/ PHLEGM	=	0.12 x POLLUTE (0.08)	-	0.057 x AVTEMP (0.0001)	+ 0.21 x BCO (0.04)
		- 1.25 x BHCX (0.004)	+	16.6 x BSO <sub>2</sub> (0.06)	
STUFFY NOSE	=	0.20 x POLLUTE (0.001)	+	0.09 x MEANCO (0.10)	- 0.05 x AVTEMP (0.0002)
		- 0.05 x MEANRBCO (0.04)			
NASAL DISCHARGE	=	0.19 x POLLUTE (0.005)	+	0.008 x SO <sub>2</sub> (0.13)	- 0.08 x AVTEMP (0.0001)
		+ 0.26 x BCO (0.01)	-	1.28 x BHCX (0.001)	
BREATHING FATIGUE	=	0.28 x POLLUTE (0.0001)	-	0.11 x MEANCO (0.02)	- 0.0048 x BSO <sub>4</sub> (0.03)
		+ 0.0048 x BNO <sub>3</sub> (0.13)			
LEG FATIGUE	=	0.11 x POLLUTE (0.15)	+	0.063 x AVTEMP (0.0001)	- 1.23 x BHCX (0.01)
		+ 20.1 x BNO <sub>2</sub> (0.02)	-	0.11 x BNO <sub>3</sub> (0.01)	
EYE IRRITATION	=	0.16 x POLLUTE (0.008)			

Gage and MOE pollutant exposures and the pollution rating scale (POLLUTE) are included in the predictor list.

Bracketed values are the p values associated with Student's t test of the hypothesis that the partial regression coefficient is zero.

For the symptom variables LEG and BREATHING FATIGUE N = 385, for all others N = 335.

The letter B preceeding the pollutant variable name represents the Breadalbane MOE site.

TABLE 13 continued:

UNITS OF MEASURE FOR VARIABLES:

See Table 9 for GAGE variables.

COH - coefficient of haze (0.1 COH/1000 ft.)

CO - (ppm)

NO, NO<sub>2</sub>, & SO<sub>2</sub> - (0.1 ppm)

API - air pollution index (unitless)

O<sub>3</sub> - ozone (ppb)

HGX - total hydrocarbons x-ray analysis (0.1 ppm)

SO<sub>4</sub> - sulphates (0.1 micrograms/m<sup>3</sup>)

NO<sub>3</sub> - nitrates (0.1 micrograms/m<sup>3</sup>)

PART - suspended particulate (HI-VOL sampler) (micrograms/m<sup>3</sup>)



TABLE 14      MULTIPLE REGRESSION EQUATIONS FOR SYMPTOMS AND EXPOSURE:  
OBTAINED FROM BACKWARD STEPWISE MULTIPLE LINEAR REGRESSION

SORE/DRY THROAT	=	-0.05 x AVTEMP (0.0001)			
COUGH	=	0.14 x MEANCO (0.002)	-	0.01 x AVRH (0.04)	- 0.053 x MEANRBCO (0.04)
SPUTUM/ PHLEGM	=	-0.05 x AVTEMP (0.0001)	+	0.20 x BCO (0.05)	- 0.95 x BHCX (0.02)
STUFFY NOSE	=	-0.03 x AVTEMP (0.007)	+	0.11 x MEANCO (0.04)	- 0.59 x BCHX (0.09)
NASAL DISCHARGE	=	-0.08 x AVTEMP (0.0001)	+	0.008 x SO <sub>2</sub> (0.13)	+ 0.026 x BCO (0.01)
		-1.30 x BHCX (0.0009)			
BREATHING FATIGUE	=	0.037 x AVTEMP (0.002)	-	0.005 x BSO <sub>2</sub> (0.02)	
LEG FATIGUE	=	0.06 x AVTEMP (0.0002)	-	0.0073 x BNO <sub>3</sub> (0.06)	
EYE IRRITATION	=	0.014 x AVTEMP (0.18)			
POLLUTE	=	0.039 x AVTEMP (0.002)	+	0.010 x NO <sub>2</sub> (0.001)	+ 0.15 x MEANCO (0.008)
		-9.85 x BNO (0.11)	-	12.5 x BSO <sub>2</sub> (0.10)	

Gage and MOE pollutant exposures are included in the predictor list.

Bracketed values are the p values associated with Student's t test of the hypothesis that the partial regression coefficient is zero.

For the symptom variables LEG and BREATHING FATIGUE N = 385, for all others N = 335.

Same abbreviations as Table 13.

TABLE 15      MULTIPLE REGRESSION EQUATIONS FOR PULMONARY FUNCTION VARIABLES AND EXPOSURES OBTAINED FROM BACKWARD STEPWISE MULTIPLE LINEAR REGRESSION

DPEF	=	0.004 x PEAKCO	-	0.016 x MEANRBCO	
		(0.18)		(0.05)	
PEFPOST	=	-0.004 x BO <sub>3</sub>			
		(0.01)			
DIC	=	0.01 x MEANRBCO			
		(0.02)			
ICPOST	=	0.007 x MEANRBCO	-	0.018 x POLLUTE	
		(0.04)		(0.03)	
DFVC	=	0.0007 x NO <sub>2</sub>	-	0.006 x BAPI	+ 0.0007 x BSO <sub>4</sub>
		(0.03)		(0.009)	(0.01)
FVCPOST	=	0.0006 x BNO <sub>3</sub>			
		(0.02)			
DFEV <sub>1</sub>	=	-0.005 x BAPI	+	0.0009 x BSO <sub>4</sub>	
		(0.05)		(0.0004)	
FEV <sub>1</sub> POST	=	-1.26 x BNO	+	0.0007 x BSO <sub>4</sub>	
		(0.008)		(0.0001)	
DFEF <sub>50</sub>	=	-4.65 x BNO <sub>2</sub>	+	0.0028 x BPART	
		(0.01)		(0.02)	
FEF <sub>50</sub> POST	=	-0.02 x MEANRBCO	-	0.016 x DRBCO	+ 0.019 x BAPI
		(0.01)		(0.01)	(0.004)
		-4.2 x BNO <sub>2</sub>			
		(0.03)			
DFEF <sub>75</sub>	=	-0.002 x NO <sub>2</sub>	+	0.01 x BAPI	- 7.26 x BSO <sub>2</sub>
		(0.01)		(0.01)	(0.0006)
FEF <sub>75</sub> POST	=	-0.002 x NO <sub>2</sub>	-	5.1 x BSO <sub>2</sub>	+ 0.002 x BSO <sub>4</sub>
		(0.004)		(0.005)	(0.0002)

Gage and MOE pollutant exposures and the pollution rating scale (POLLUTE) are included in the predictor list.

Bracketed values are the p values associated with Student's t test of the hypothesis that the partial regression coefficient is zero.

For all analyses N = 426.

Same abbreviations as Tables 11 and 13.

TABLE 16      MULTIPLE REGRESSION EQUATIONS FOR PULMONARY FUNCTION  
VARIABLES AND EXPOSURES OBTAINED FROM BACKWARD STEPWISE  
MULTIPLE LINEAR REGRESSION

DPEF	=	-0.043 x MEANCO	+	0.088 x PEAKCO		
		(0.03)		(0.02)		
PEFPOST	=	-0.004 x BO <sub>3</sub>				
		(0.01)				
DIC	=	0.01 x MEANRBCO				
		(0.02)				
ICPOST	=	0.009 x MEANRBCO	+	0.006 x DRBCO		
		(0.02)		(0.06)		
DFVC	=	0.0006 x NO <sub>2</sub>	-	0.0061 x BAPI	+	0.006 x BSO <sub>4</sub>
		(0.06)		(0.01)		(0.02)
FVCPOST	=	0.0006 x BNO <sub>3</sub>				
		(0.02)				
DFEV <sub>1</sub>	=	0.0005 x BSO <sub>4</sub>				
		(0.002)				
FEV <sub>1</sub> POST	=	-1.01 x BNO	+	0.0007 x BSO <sub>4</sub>		
		(0.02)		(0.0001)		
DFEF <sub>50</sub>	=	0.007 x AVTEMP				
		(0.05)				
FEF <sub>50</sub> POST	=	-0.02 x MEANRBCO	-	0.016 x DRBCO	+	0.013 x BAPI
		(0.02)		(0.02)		(0.02)
DFEF <sub>75</sub>	=	-0.002 x NO <sub>2</sub>	+	0.014 x BAPI	-	7.5 x BSO <sub>2</sub>
		(0.01)		(0.005)		(0.0004)
FEF <sub>75</sub> POST	=	-0.002 x NO <sub>2</sub>	-	4.69 x BSO <sub>2</sub>	+	0.001 x BSO <sub>4</sub>
		(0.02)		(0.01)		(0.001)

Gage and MOE pollutant exposures are included in the predictor list.

Bracketed values are the p values associated with Student's t test of the hypothesis that the partial regression coefficient is zero.

For all analyses N = 434.

Same abbreviations as Tables 11 and 13.

TABLE 17      MULTIPLE REGRESSION EQUATIONS FOR FOUR MEASURES OF BLOOD CARBON MONOXIDE AND POLLUTANT EXPOSURES OBTAINED FROM BACKWARD STEPWISE MULTIPLE LINEAR REGRESSION

$$\begin{array}{lcl} \text{RBCOPRE} & = & 0.02 \times \text{SO}_2 + 0.60 \times \text{MEANCO} - 0.043 \times \text{PEAKCO} \\ (\text{N}=564) & & (0.13)^* \quad (0.0003) \quad (0.16) \end{array}$$

$$\begin{array}{lcl} \text{RBCOPOST} & = & 0.02 \times \text{NO}_2 + 0.80 \times \text{MEANCO} - 0.051 \times \text{PEAKCO} \\ (\text{N}=565) & & (0.0004) \quad (0.0001) \quad (0.01) \end{array}$$

$$\begin{array}{lcl} \text{MEANRBCO} & = & 0.014 \times \text{NO}_2 + 0.70 \times \text{MEANCO} - 0.042 \times \text{PEAKCO} \\ (\text{N}=564) & & (0.03) \quad (0.0001) \quad (0.06) \end{array}$$

$$\begin{array}{lcl} \text{DIFFRBCO} & = & 0.018 \times \text{NO}_2 - 0.019 \times \text{SO}_2 + 0.201 \times \text{MEANCO} \\ (\text{N}=564) & & (0.03) \quad (0.04) \quad (0.11) \end{array}$$

The predictor list includes the pollution rating scale (POLLUTE).

RBCOPRE      -    Rebreathe carbon monoxide (bag concentration in ppm) taken just before the run.

RBCOPOST     -    Rebreathe carbon monoxide (bag concentration in ppm) taken immediately after the run.

MEANRBCO     -    The mean of the pre and post rebreathe readings.

DIFFRBCO     -    The difference between the pre and post rebreathe readings (RBCOPOST - RBCOPRE)

\*    The bracketed values are the p values associated with Student's t test of the hypothesis that the regression coefficient is zero.

See Table 9 for COHb% calculation.

TABLE 18      MULTIPLE REGRESSION EQUATIONS FOR FOUR MEASURES OF BLOOD CARBON MONOXIDE AND POLLUTANT EXPOSURES OBTAINED FROM BACKWARD STEPWISE MULTIPLE LINEAR REGRESSION

RBCOPRE (N=572)	=	0.01 x PART (0.13)*	+	0.48 x MEANCO (0.0005)	
RBCOPOST (N=573)	=	0.022 x NO <sub>2</sub> (0.0003)	+	0.79 x MEANCO (0.0001)	- 0.048 x PEAKCO (0.01)
MEANRBCO (N=572)	=	0.015 x NO <sub>2</sub> (0.03)	+	0.69 x MEANCO (0.0001)	- 0.042 x PEAKCO (0.045)
DIFFRBCO (N=572)	=	0.017 x NO <sub>2</sub> (0.03)	-	0.013 x SO <sub>2</sub> (0.15)	+ 0.194 x MEANCO (0.12)

The predictor list does not include the pollution rating scale (POLLUTE)

\* The bracketed values are the p values associated with Student's t test of the hypothesis that the regression coefficient is zero.

Same abbreviations as Tables 9 and 17.

TABLE 19 MULTIPLE LINEAR REGRESSION MODEL FOR POLLUTION RATING AGAINST MEAN NO<sub>2</sub>, TEMPERATURE AND CARBON MONOXIDE CONCENTRATION \*\*

	REGRESSION COEFFICIENT	STANDARD ERROR	P * VALUE	CUMULATIVE R SQUARE	PARTIAL R SQUARE
NO <sub>2</sub>	0.009	0.003	0.001	0.380	0.045
AVERAGE TEMPERATURE	0.049	0.009	0.0001	0.404	0.039
MEAN CO	0.134	0.046	0.004	0.414	0.017

\* The P values associated with Student's t test of the hypothesis that the regression coefficient is zero in the final model that has all three variables.

\*\* The NO<sub>2</sub> and the CO concentrations and temperature were measured by the Gage monitors on bicycles that accompanied the runners along their route.

Number of runners = 65 and number of runner days = 545.

TABLE 20 MULTIPLE LINEAR REGRESSION MODEL FOR POLLUTION RATING AGAINST  
MEAN NO<sub>2</sub>, TEMPERATURE AND REBREATHE CO CONCENTRATION \*\*

	REGRESSION COEFFICIENT	STANDARD ERROR	P * VALUE	CUMULATIVE R SQUARE	PARTIAL R SQUARE
NO <sub>2</sub>	0.011	0.003	0.0001	0.380	0.045
AVERAGE TEMPERATURE	0.040	0.009	0.0001	0.404	0.039
MEANRBCO	0.038	0.019	0.046	0.409	0.008

\* The P values associated with Student's t test of the hypothesis that the regression coefficient is zero in the final model that has all three variables.

\*\* The NO<sub>2</sub> concentration and temperature were measured by the Gage monitors on bicycles that accompanied the runners along their route.

MEANRBCO - Mean of the pre and post run rebreathe CO, bag concentration (ppm).

The number of runners = 65 and the number of runner days = 545.

TABLE 21      MULTIPLE LINEAR REGRESSION MODEL FOR POLLUTION RATING AGAINST  
MEAN NO<sub>2</sub>, RESPIRABLE PARTICULATE AND OZONE CONCENTRATION \*\*

	REGRESSION COEFFICIENT	STANDARD ERROR	P * VALUE	CUMULATIVE R SQUARE	PARTIAL R SQUARE
NO <sub>2</sub>	0.011	0.003	0.0001	0.380	0.045
OZONE BREADALBANE	0.012	0.003	0.0001	0.399	0.032
RESPIRABLE PARTICULATES	0.004	0.002	0.019	0.406	0.011

\* The P values associated with Student's t test of the hypothesis that the regression coefficient is zero in the final model that has all three variables.

\*\* The NO<sub>2</sub> and respirable particulate concentrations were measured by the Gage monitors on bicycles that accompanied the runners along their route.

The number of runners = 65 and the number of runner days = 545.



TABLE 22      FREQUENCY DISTRIBUTION OF THE SLOPES OF THE INDIVIDUAL  
 RUNNERS - POLLUTE AGAINST MEAN NO<sub>2</sub> \*

RANGE OF SLOPES		FREQUENCY
-0.04	to -0.02	3
-0.02	to 0.00	15
0.00	to 0.02	16
0.02	to 0.04	14
0.04	to 0.06	2
0.06	to 0.08	4
0.08	to 0.10	1
0.10	to 0.12	2
0.12	to 0.14	0
0.14	to 0.16	<u>1</u>
		58

MEAN = 0.021      STD DEV = 0.037      MEDIAN = 0.011

P VALUE FOR WILCOXON SIGNED RANK TEST = 0.00004

90% RANGE      -0.02      to      0.11

Number of positive/negative slopes = 40/18

P value for the sign test = 0.004

\* Only slopes based on three or more days of measurements  
 are reported in this table.

POLLUTE is a subjective rating scale of pollution that can  
 assume values from 0 - no pollution - to 10 - severe  
 pollution.

TABLE 23      FREQUENCY DISTRIBUTION OF THE SLOPES OF THE INDIVIDUAL  
 RUNNERS - POLLUTE AGAINST MEAN CARBON MONOXIDE

RANGE OF SLOPES			FREQUENCY
-1.25	to	-1.00	1
-1.00	to	-0.75	0
-0.75	to	-0.50	2
-0.50	to	-0.25	3
-0.25	to	0.00	13
0.00	to	0.25	24
0.25	to	0.50	11
0.50	to	0.75	2
0.75	to	1.00	1
1.00	TO	1.25	1
1.25	to	1.50	<u>1</u>
			59

MEAN = 0.10      STD DEV = 0.38      MEDIAN = 0.075

P VALUE FOR WILCOXON SIGNED RANK TEST = 0.02

90% RANGE      -0.51      to      0.77

Number of positive/negative slopes = 40/19

P value for the sign test = 0.006

\* Only slopes based on three or more days of measurements  
 are reported in this table.

POLLUTE is a subjective rating scale of pollution that can  
 assume values from 0 - no pollution - to 10 - severe  
 pollution.

TABLE 24      FREQUENCY DISTRIBUTION OF THE SLOPES OF THE INDIVIDUAL  
 RUNNERS - POLLUTE AGAINST MEAN TEMPERATURE

RANGE OF SLOPES		FREQUENCY
-0.5	to -0.4	1
-0.4	to -0.3	0
-0.3	to -0.2	0
-0.2	to -0.1	2
-0.1	to 0.0	15
0.0	to 0.1	25
0.1	to 0.2	11
0.2	to 0.3	3
0.3	to 0.4	1
0.4	to 0.5	<u>1</u>
		59

MEAN = 0.058      STD DEV = 0.129      MEAN = 0.044

P VALUE FOR WILCOXON SIGNED RANK TEST = 0.0001

90% RANGE      -0.10      to      0.29

Number of positive/negative slopes = 41/18

P Value for the sign test = 0.004

\* Only slopes based on three or more days of measurements  
 are reported in this table.

POLLUTE is a subjective rating scale of pollution that can  
 assume values from 0 - no pollution - to 10 - severe  
 pollution.

TABLE 25 FREQUENCY DISTRIBUTION OF THE SLOPES OF THE INDIVIDUAL RUNNERS - RBCOPOST AGAINST MEAN CARBON MONOXIDE

RANGE OF SLOPES		FREQUENCY
-1.50	to -1.00	1
-1.00	to -0.50	1
-0.50	to 0.00	7
0.00	to 0.50	15
0.50	to 1.00	15
1.00	to 1.50	12
1.50	to 2.00	2
2.00	to 2.50	2
> 2.50		<u>1</u> 57

MEAN = 0.88      STD DEV = 1.50      MEDIAN = 0.71

P VALUE FOR WILCOXON SIGNED RANK TEST = 0.00000007

90% RANGE      -0.41      to      2.42

Number of positive/negative slopes = 48/9

P value for the sign test < 0.0001

\* Only slopes based on three or more days of measurements are reported in this table.

RBCOPOST is an estimate of a runner's blood carbon monoxide level measured at the end of the run.

TABLE 26 FREQUENCY DISTRIBUTION OF THE SLOPES OF THE INDIVIDUAL RUNNERS - RBCOPOST AGAINST MEAN NO<sub>2</sub>

RANGE OF SLOPES		FREQUENCY
LESS THAN	-0.1	4
-0.1	to 0.0	11
0.0	to 0.1	33
0.1	to 0.2	5
0.2	to 0.3	1
GREATER THAN	0.3	<u>3</u>
		57

MEAN = 0.031      STD DEV = 0.127      MEDIAN = 0.033

P VALUE FOR WILCOXON SIGNED RANK TEST = 0.0005

90% RANGE      -0.11      to      0.29

Number of positive/negative slopes = 42/15

P value for the sign test = 0.0006

\* Only slopes based on three or more days of measurements are reported in this table.

RBCOPOST is an estimate of a runner's blood carbon monoxide level measured at the end of the run.

TABLE 27      MULTIPLE LINEAR REGRESSION MODELS FOR THE SUBJECTIVE POLLUTION RATING SCALE (POLLUTE) AGAINST MEAN NO<sub>2</sub>, CARBON MONOXIDE AND TEMPERATURE

	REGRESSION COEFFICIENT	STANDARD ERROR	T RATIO	P VALUE
<u>MODEL A</u>				
NO <sub>2</sub> - GAGE	0.0086	0.0027	3.15	0.0017
TEMPERATURE - GAGE	0.0476	0.0093	5.13	0.0001
CO - GAGE	0.1235	0.0457	2.70	0.0071

<u>MODEL B</u>				
NO <sub>2</sub> - BREADALBANE	3.11	4.88	0.64	0.52
TEMPERATURE - GAGE	0.0519	0.0095	5.49	0.0001
CO - GAGE	0.16	0.048	3.36	0.0009

<u>MODEL C</u>				
NO <sub>2</sub> - GAGE	0.012	0.003	4.41	0.0001
TEMPERATURE - GAGE	0.038	0.0085	4.43	0.0001
CO - BREADALBANE	-0.084	0.084	-1.01	0.31

<u>MODEL D</u>				
NO <sub>2</sub> - BREADALBANE	11.52	4.99	2.31	0.021
TEMPERATURE - GAGE	0.0386	0.0087	4.46	0.0001
CO - BREADALBANE	-0.069	0.091	-0.76	0.45

NO<sub>2</sub> - GAGE and CO - GAGE are the measurements of NO<sub>2</sub> and carbon monoxide concentrations using the portable monitors transported on the bicycles.

NO<sub>2</sub> - BREADALBANE and CO - BREADALBANE are the measurements of NO<sub>2</sub> and carbon monoxide concentrations made by the Ministry of the Environment at their Breadalbane location.

### Publications, Presentations and Reports Related to the Study

Appendices IV - VIII contain the progress reports submitted to the Ministry of the Environment.

A poster presentation was made at both the 1987 and 1988 Technology Transfer Conference (Appendix IX and X).

A poster presentation was made at the University of Toronto ("U. of T. day"), October 24, 1987 and October 15, 1988.

Four abstracts were submitted to the 1986-1989 Technology Transfer Conferences (Appendix XI - XIV), and extended abstracts in 1987 (Appendix XII) and 1988 (Appendix XIII). An abstract was submitted and accepted for the Las Vegas symposium on human health November 27-30, 1989 (Appendix XV).

A chapter entitled "Air Pollution and Exercise" in Current Therapy in Sports Medicine is in press.

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